PREDICTIVE VALUE OF CORD BILIRUBIN IN NEONATAL HYPERBILIRUBINEMIA

Dissertation submitted in partial fulfilment of the

Requirement for the award of the Degree of

M.D. DEGREE – BRANCH VII

PAEDIATRICS

APRIL 2016

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY,

CHENNAI,

TAMIL NADU

CERTIFICATE

This is to certify that the Dissertation entitled "**PREDICTIVE VALUE OF CORD BILIRUBIN IN NEONATAL HYPERBILIRUBINEMIA**" submitted by Dr.M.Deepa, MBBS., DCH., to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfilment for the award of M.D (Paediatrics) is a bonafide work carried out by her under my guidance and supervision during the academic year 2014-2016. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other.

GUIDE
Dr.A.S.Babu Kandakumar, MD.,DCH.,DNB.,MNANS.,
Department Paediatrics,
Tirunelveli Medical College,
Tirunelveli- 627011.

HOD
Prof.Dr.C.Krishnamurthy,MD.,(Paediatrics).,
Department Paediatrics,
Tirunelveli Medical College,
Tirunelveli- 627011.

Dr K.Sithy Athiya Munavarah, DCH., MD(PATHO)
THE DEAN,
Tirrunelveli Medical College,
Tirunelveli- 627011.

DECLARATION

I, Dr.M.Deepa, MBBS., DCH., solemnly declare that the Dissertation titled **"PREDICTIVE VALUE OF CORD BILIRUBIN IN NEONATAL HYPERBILIRUBINEMIA"** had been prepared by me under the expert guidance and supervision of **Dr.A.S.Babu Kandakumar, MD.,DCH.,** Professor, Department of Paediatrics, Tirunelveli Medical College Hospital, Tirunelveli.

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the regulation for the award of M.D. Degree (Branch VII) in Paediatrics.

It was not submitted to the award of any degree/diploma to any University either in part or in full previously.

Place: Tirunelveli.
Date:

Dr.M.Deepa, MBBS., DCH.,
POST GRADUATE,
M.D. (Paediatrics),
Tirunelveli Medical College,
Tirunelveli.

ACKNOWLEDGEMENT

I am very much thankful to the Dean **Dr K.Sithy Athiya Munavarah,DCH., MD(PATHO)**, Triunelveli Medical College Hospital, Tirunelveli, who has granted permission to do this study in this institution,

I take this opportunity to express my deepest sense of gratitude to professor **Prof.Dr.C.Krishnamurthy,MD.,(Paediatrics).,** Head of the Department of Paediatrics, Tirunelveli Medical College Hospital, Tirunelveli for encouraging me and rendering timely suggestions and guiding me throughout the course of this study. I will be forever indebted to her for her constant support.

I sincerely thank my professor **Dr.T.R.R. Ananthy Shri, M.D., (Paediatrics)., Dr.M.Mathivanan,M.D.,(DCH).,** for their support and guidance.

I am very much thankful to professor **Dr.Meena,MD., DGO., DNB**, Head of Department of Obstetrics and Gynaecology for providing valuable support and guiding through the study.

I am extremely thankful to my guide **Dr.A.S.Babu Kandakumar, MD.,DCH.,** for guiding me throughout the study.

I am extremely thankful to all my **Assistant Professors** of the **Department of Paediatrics** for their guidance and support throughout my study period in this institution.

I thank **Prof. P. Arumugam** statistician for their useful inputs.

I wish to express my gratitude to my parents, my sisters, my husband and my son for their support throughout my study.

I also like to express my gratitude to my friends and colleagues who have always been a source of love, support and encouragement.

Last but not least, I am very much thankful to all antenatal mothers of Tiruneveli Medical College without whom this study would not have been possible.

CONTENTS

TIRUNELVELI MEDICAL COLLEGE

INSTITUTIONAL RESEARCH ETHICS COMMITTEE
TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462-2572733-EXT; 91-462-2572944; 91-462-2579785; 91-462-2572611-16
online@tvmc.ac.in, tirec@tvmc.ac.in; www.tvmc.ac.in

CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO: 671/PAED/ 2015

PROTOCOL TITLE: PREDICTIVE VALUE OF UMBLICAL CORD BILIRUBIN IN NEONAL HYPERBILIRUBIN.

PRINCIPAL INVESTIGATOR: DR.M. DEEPA, MBBS.,DCH.,

DESIGNATION OF PRINCIPAL INVESTIGATOR POST GRADUATE IN PAEDIATRICS
DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE , TIRUNELVELI

Dear , Dr.M.Deepa, MBBS.,Dch., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 09.02.15.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

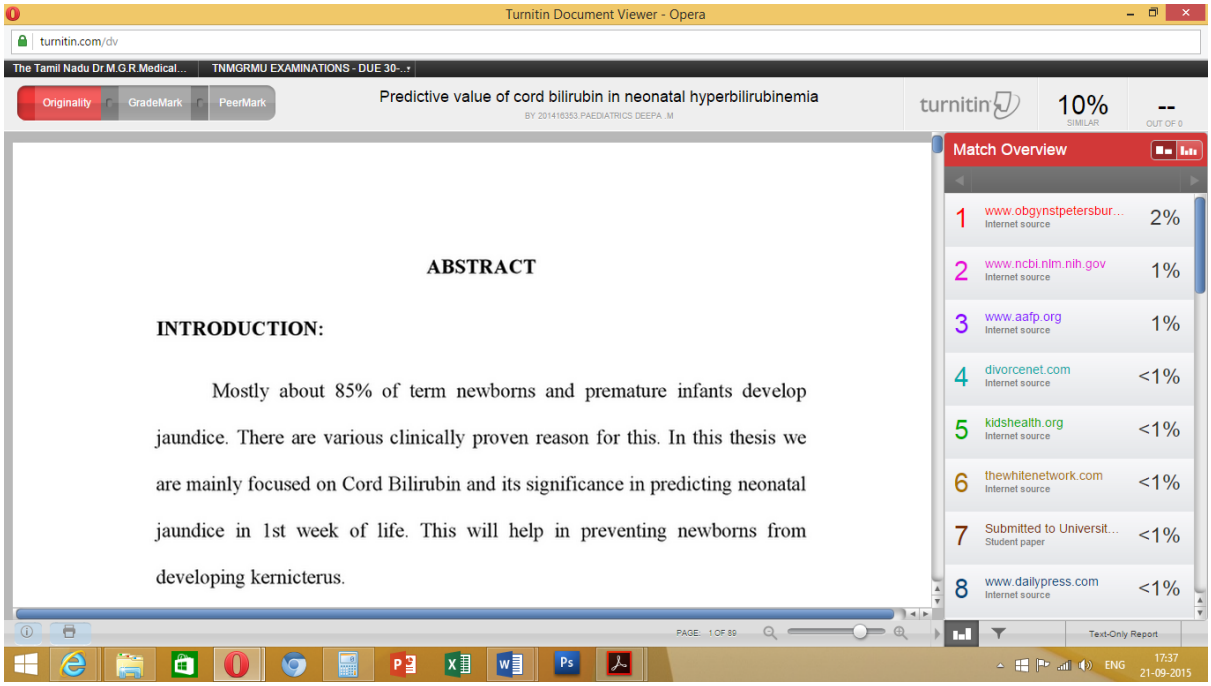
THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

Dr.K.Shantaraman MD
Registrar, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

Dr.V.Ramasubramanian MD DM
Member Secretary, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India


Turnitin Document Viewer - Opera
turnitin.com/dv
The Tamil Nadu Dr.M.G.R.Medical...
TNMGRMU EXAMINATIONS - DUE 30-...
Originality
GradeMark
PeerMark
Predictive value of cord bilirubin in neonatal hyperbilirubinemia
BY 201416353.PAEDIATRICS DEEPA .M
turnitin
10%
SIMILAR
--
OUT OF 0
ABSTRACT
INTRODUCTION:
Mostly about 85% of term newborns and premature infants develop jaundice. There are various clinically proven reason for this. In this thesis we are mainly focused on Cord Bilirubin and its significance in predicting neonatal jaundice in 1st week of life. This will help in preventing newborns from developing kernicterus.
Match Overview
1 www.obgynstpetersbur... Internet source 2%
2 www.ncbi.nlm.nih.gov Internet source 1%
3 www.aafp.org Internet source 1%
4 divorcenet.com Internet source <1%
5 kidshealth.org Internet source <1%
6 thewhitenetwork.com Internet source <1%
7 Submitted to Universit... Student paper <1%
8 www.dailypress.com Internet source <1%
PAGE: 1 OF 89
Text-Only Report
ENG
17:37
21-09-2015

ABBREVIATION

AGA	-	Appropriate for Gestational Age
CBR	-	Cord Bilirubin
CPD	-	Cephalo Pelvic Dislocation
CS	-	Caesarean Section
FFP	-	Fresh Frozen Plasma
GA	-	Gestational Age
G6PD	-	Glucose-6-Phosphate Dehydrogenase
EDD	-	Expected Date of Delivery
Hb	-	Haemoglobin
HBR	-	Hyperbilirubinemia
HDN	-	Haemolytic Disease of Newborn
IVIG	-	Intravenous Immunoglobulin
LBW	-	Low Birth Weight
LMP	-	Last Menstrual Period
LN	-	Labor Natural
NHBR	-	Non Hyperbilirubinemia
NPV	-	Negative Predictive Value
PDA	-	Patent Ductus Arteriosis
PIH	-	Pregnancy Induced Hypertension
PPV	-	Positive Predictive Value
SBR	-	Serum Bilirubin
TCB	-	Transcutaneous Bilirubinometer
T/ID	-	Total/ Indirect Bilirubin
TSB	-	Total Serum Bilirubin
UDPGT	-	Uridine Di Phosphate Glucuronosyl Transferase

ABSTRACT

INTRODUCTION:

Mostly about 85% of term newborns and premature infants develop jaundice. There are various clinically proven reason for this. In this thesis we are mainly focused on Cord Bilirubin and its significance in predicting neonatal jaundice in 1st week of life. This will help in preventing newborns from developing kernicterus.

AIM OF THE STUDY:

To evaluate the predictive value of umbilical cord bilirubin in identifying term newborns with ABO /Rh incompatibility for subsequent hyperbilirubinemia in 1st week of life.

METHOD OF STUDY:

Prospective clinical study in Tiruneveli Medical College Hospital (TVMCH) which is carried out in all consecutive term newborns with ABO /Rh incompatibility over a period of 8 months (February 2015 to SePhototherapy ember 2015) duration.

The study population was initially followed up clinically by Kramer's method and by transcutaneous bilirubinometer. Newborns identified with jaundice were followed up using serial serum bilirubin values.

METHOD OF ASSAY - SERUM BILIRUBIN:

Total serum bilirubin, conjugated bilirubin, unconjugated bilirubin were obtained via the calorimetric diazo method.

RESULTS OF STUDY:

Female babies were found to be higher i.e.,62.7% when compared to male babies 37.% Rh incompatibility is statistically significantly associated with PHOTOTHERAPY . Mean total CORD BILIRUBIN is more in Rh incompatibility (4.23) when compared to ABO incompatibility (3.86). Mother's blood group has statistical signification with HYPERBILIRUBINEMIA and NHYPERBILIRUBINEMIA but it was not seen in baby's blood group. The mean Hb of HYPERBILIRUBINEMIA and NHYPERBILIRUBINEMIA is 15.6 ± 0.7 and 14.2 ± 2 gm/dL respectively and difference between them was statistically significant. The mean CORD BILIRUBIN between babies with HYPERBILIRUBINEMIA and NHYPERBILIRUBINEMIA was 4 ± 0.5 and 2.3 ± 0.3 mg/dL respectively and difference between them was statistically significant. The relationship between total CORD BILIRUBIN and SBR was statistically significant

CONCLUSION

Study concludes that the total CORD BILIRUBIN in healthy term newborns provides prediction for neonatal jaundice in 1st week of life. The cut off value being 3.25 with 96.0% of specificity and 96.0% sensitivity. It is also evident that the presence of incompatibility in newborns (ABO, Rh) was statistically significant for occurrence of high total CORD BILIRUBIN that indicates PHOTOTHERAPY treatment.

KEYWORDS

AGA	-	Appropriate for Gestational Age
CBR	-	Cord Bilirubin
CPD	-	Cephalo Pelvic Dislocation
CS	-	Caesarean Section
FFP	-	Fresh Frozen Plasma
GA	-	Gestational Age
G6PD	-	Glucose-6-Phosphate Dehydrogenase
EDD	-	Expected Date of Delivery
Hb	-	Haemoglobin
HBR	-	Hyperbilirubinemia
HDN	-	Haemolytic Disease of Newborn
IVIG	-	Intravenous Immunoglobulin
LBW	-	Low Birth Weight
LMP	-	Last Menstrual Period
LN	-	Labor Natural
NHBR	-	Non Hyperbilirubinemia
NPV	-	Negative Predictive Value
PDA	-	Patent Ductus Arteriosis
PIH	-	Pregnancy Induced Hypertension
PPV	-	Positive Predictive Value
SBR	-	Serum Bilirubin
TCB	-	Transcutaneous Bilirubinometer
T/ID	-	Total/ Indirect Bilirubin
TSB	-	Total Serum Bilirubin
UDPGT	-	Uridine Di Phosphate Glucuronosyl Transferase

INTRODUCTION

Babies are called newborns during their first month of life. After birth mostly we can witness that babies sleep a lot this is because a lot of changes that takes place inside their body system. Mostly about 85% of term newborns and premature infants develop jaundice. There are various clinically proven reason for this. In this thesis we are mainly focused on Cord Bilirubin and its significance in predicting neonatal jaundice. Most babies have mild jaundice. It usually gets better or goes away on its own within a week or two without causing problems. Generally jaundice should be taken seriously, it may be physiological or pathological. In rare cases, if the bilirubin level stays high and isn't treated, it can cause brain damage called kernicterus (This term was introduced in early 1900s, it refers to the yellow staining of basal ganglia that was noticed in newborns died with severe jaundice). This can lead to serious lifetime problems too. At times this condition becomes life threatening if it's not properly taken care which may lead to hyperbilirubinemia. We are mainly concerned about various causes, ill effects as well the prevention mechanism of it.

DEFINITION

Jaundice occurs because your baby's body has more bilirubin level than it can get rid of. Bilirubin is a yellow (pigment) substance that's made when the body breaks down old red blood cells (RBC's).Generally the life span of RBC is 120days in adults and whereas the life span of newborn RBCs are comparatively lesser. Newborns RBCs are higher in number and shorter in span. It’s mainly excreted from the body through urine and stool. When a mother is pregnant, her body removes bilirubin from her babies through the placenta. After birth, your baby's body must get rid of this bilirubin on its own. In most cases, babies have what's called physiologic jaundice. It occurs because their organs aren't yet able to get rid of this excess bilirubin on their own. This type of jaundice usually appears about 24 hours after birth. It at times gets worse by the third or fourth day, and then it goes away in about a week. Many babies get jaundice (hyperbilirubinemia) in their first few days of life. In this condition the skin and the whites of a baby's eyes appear yellow because of excess bilirubin in the blood.

In rare cases, jaundice may be caused by other things, such as an infection, a problem with the baby's digestive system, or a problem with the moms and baby's blood groups or types (ABO/Rh incompatibility). Your baby may have one of these problems if jaundice appears less than a day after birth. It's always better to check with the levels and take precautionary measures. In a normal adult serum bilirubin level ranges from ≤1mg/dL. Whereas adult's develop jaundice if the level of serum bilirubin ≥2mg/dL. The same condition

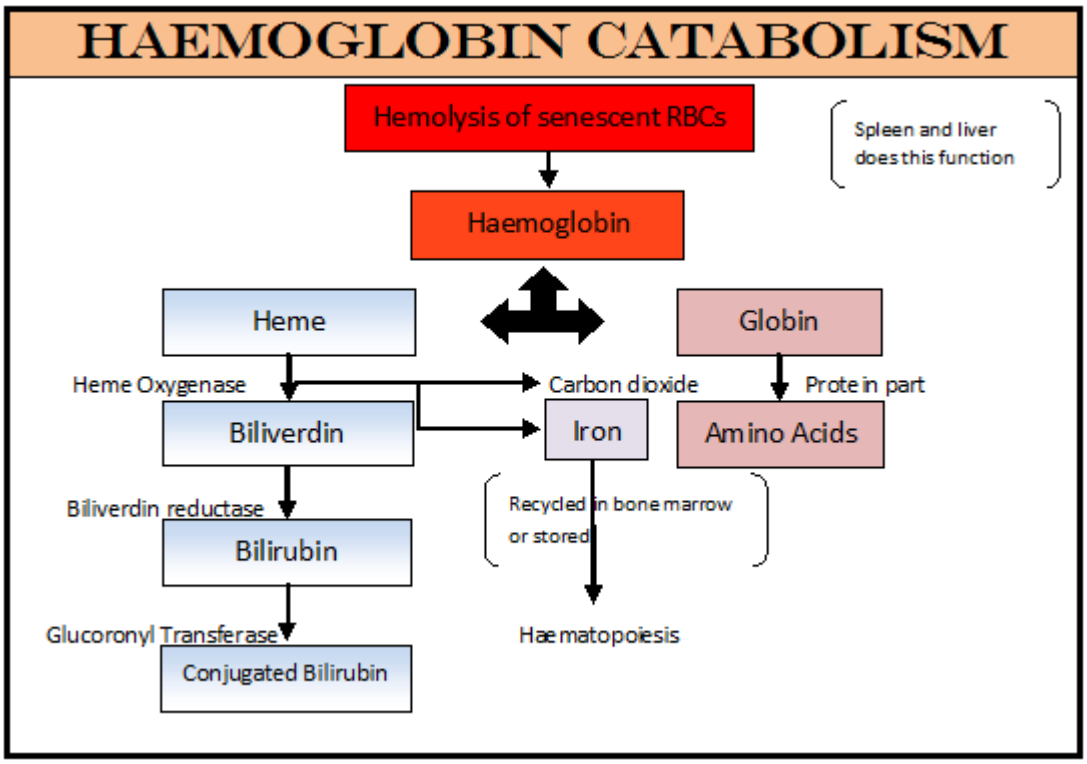
when compared to newborns where the serum bilirubin levels ≥7mg/dL they develop jaundice. Generally 6.1% of well term newborns have serum bilirubin levels ≥12.9mg/dL.

EPIDEMIOLOGY

Jaundice a most common condition that requires medical attention. In order to develop a diagnostic as well management of jaundice in newborns, we need to have a clear back ground idea about pathological and non pathological factors as well their bilirubin levels. Approximately 60-65% of term infants and 80-85% of preterm infants develop jaundice. About 10% of breast fed babies still have been identified with jaundice within 1month of age. Various risk factors that are responsible are Birth weight less than 2500gms or premature infants, if previous siblings TSB $\geq$ 12 mg/dL subsequent sibling is prone to jaundice, newborns of mother who have diabetics Section, male child have been reported more compared to females, People living at higher altitudes, East Asian babies have increased bilirubin compared to others and neonatal infection adds risk to hyperbilirubinemia.

ETIOLOGY

Jaundice is generally caused by the excess amount of bilirubin. The RBCs Section present in blood help in transport of oxygen and carbon-di-oxide. Generally the life span of RBCs in adult 120 days and infants 70-90days.



Senescent RBCs Section break down during which the protein part globulin produce bilirubin as end product. Various cause of jaundice have been listed as follows:

PHYSIOLOGICAL JAUNDICE:

Various factors that bring about physiological jaundice are increased RBC volume/kg and reduced RBCs Section life span in newborns, increased ineffective erythropoiesis and increased non haemoglobin turnover, increased enterohepatic circulation due to increased intestinal β-glucuronidase, decreased

uptake of bilirubin from plasma, decreased conjugation due to decreased UDPGT activity and decreased excretion of bilirubin by liver.

NON PHYSIOLOGICAL JAUNDICE:

Various factors that bring about non physiological jaundice were as follows:

Jaundice occurring ≤ 24 hours of age

Rise of serum bilirubin ≥ 0.2 mg/dL/hr

Associated with features of sepsis

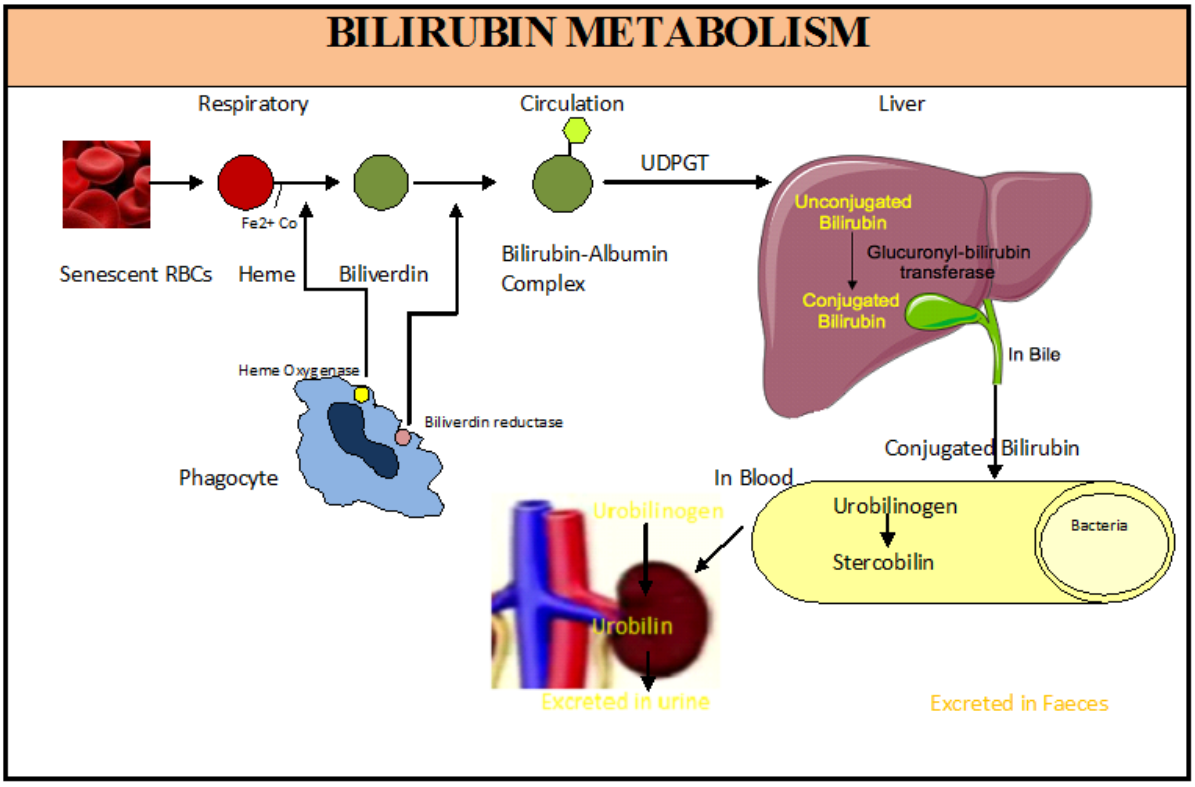
Jaundice more than 8 days in term and more than 14 days in preterm

CAUSES:

Sepsis, trauma, TORCH infection, hypothyroidism and infant of diabetic mother.

BILIRUBIN METABOLISM

A normal newborn produces 6-10mg of bilirubin/kg/day where as adults 3-4mg/kg/day. RBC contains Heme and globin. The senescent RBC releases Heme in reticuloendothelial system i.e., the major source of 75% bilirubin. 1gm of haemoglobin equal to 34mg of bilirubin. It's found in the blood stream as two forms indirect (Unconjugated) bilirubin and direct (conjugated) bilirubin. Accelerated release produces hyperbilirubinemia. The balance 25% of bilirubin is called Early-labelled bilirubin, which is derived from haemoglobin released by ineffective erythropoiesis in bone marrow from other heme proteins in tissue and from free heme.



In the senescent RBC haemoglobin is broken into Heme and globin. The heme is degraded by heme oxygenase, which releases iron and form carbon

monoxide and biliverdin. This biliverdin is then reduced to bilirubin by biliverdin reductase. This forms a Bilirubin-Albumin complex. This complex is broken down by UDPGT. Further bilirubin enter the liver and gets modified as excretable conjugated form in the presence of Glucoronyl-bilirubin transferase enzyme. The conjugated bilirubin enters the intestine for excretion as Urobilinogen and Stercobilin, at times it may be deconjugated by certain strains of bacteria and bilirubin may re-enter the circulatory path. Circumstances like increased production of bilirubin, increased enterohepatic circulation, poor uptake of liver, enzyme deficit for conjugation (UDPGT- Uridine DiPhosphate Glucuronosyl Transferase which is required for conjugation of bilirubin), babies who are not fed well with breast milk also were found to have higher risk of hyperbilirubinemia, on the other hand breast milk fed babies were found to have low levels of intestinal bacteria. Mainly the bilirubin is removed from the body through faeces (Stool) as Stercobilin and urine as urobilin which gives the colour to faeces and urine.

TYPES OF JAUNDICE

The most common types of jaundice are as follows:

PHYSIOLOGICAL (NORMAL) JAUNDICE:

Most newborns have this mild jaundice because their liver is still in the phase of maturing. It appears when a baby 2 to 4 days old and disappears by 1 to 2 weeks of age (≥ 2 mg/dL). In full term infants peak occurs 6-8mg/dL by 3 to 5 days of age and then falls (≥ 12mg/dL). In premature infants the rise may be 10-12mg/dL on 5th day of life (rise in ≥ 15mg/dL). In newborns unconjugated bilirubin is not excreted much, whereas the conjugated bilirubin excretion is also limited. As a result high serum bilirubin concentration leads to physiological jaundice.

NON PHYSIOLOGICAL JAUNDICE:

Jaundice ≤ 24 hours of age. Rise of serum bilirubin that requires phototherapy. Rise of serum bilirubin ≥ 0.2 mg/dL/hour. It's associated with features of sepsis. Jaundice more than 8days in term newborn or more than 14days in preterm newborn.

BREASTFEEDING JAUNDICE:

Jaundice occurs when breastfeeding babies don't get enough breast milk from their mother or difficulty in breast feeding persist. It occurs after day 3 of life and usually the peak level is more than 12mg/dL in 12 to 13% of breast feeding babies. The main factor responsible for breast feeding jaundice is

decreased intake of breast milk leads to late bilirubin elimination and increased enterohepatic circulation.

BREAST MILK JAUNDICE:

This jaundice usually has a late on set and the incident varies form 2-4%. In day 4 instead of decrease in serum bilirubin the level may rise to 20-30mg/dL by 14days. This may return to normal by 4 to 12 weeks of age. The various mechanism responsible for this are unidentified factors that interferes with bilirubin metabolism and increased enterohepatic circulation (increased β-glucuronidase) leads to decreased intestinal bacteria that converts conjugated bilirubin to Urobilinoids.

RISK FACTORS OF JAUNDICE

Several risk factors have been identified for the development of severe hyperbilirubinemia in newborns, they have been listed as follows:

Low gestational age.

Exclusive breast feeding baby with weight loss or inadequate feeding habits. Visible jaundice in ≤ 24 hours of age.

Isoimmune or other haemolytic diseases.

Elder sibling with jaundice. Shorter gestation less than 38 weeks.

Maternal age greater than 25 years.

Visible Bruising or Cephalohematoma.

CLINICAL FEATURES AND EXAMINATION

Baby appears yellowish tinged in skin and eyes according to level of bilirubin. According to its age, severity and onset it's further divided into the following:

EARLY STAGE:

It occurs between days 1 to 2 and it's very uncommon. It may be because of ABO, Rh, etc.

ACUTE STAGE:

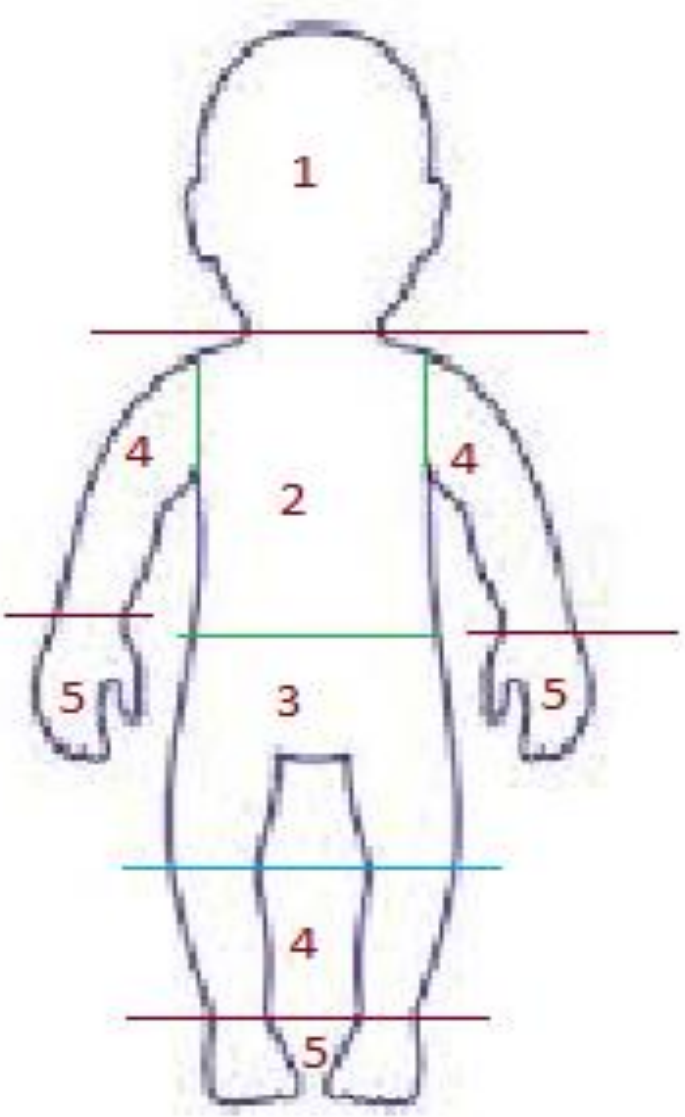
This condition is severe and of sudden onset between 3-10 days. They don't last for longer duration and conditions like poor suckling, high pitched cry, lethargy and decreased tone. This is followed by increased tone of extensor muscle, fever, irritability and seizure. Onset of these conditions followed by opisthotonus, shrill cry, apnea, seizures and leads to death.

CHRONIC STAGE:

This is a condition developed over a longer period of time, days 14 plus. They need proper medical attention for appearances like Athetosis, sensory neural deafness, upward gaze defect, dental dysplasia and intellectual defects.

KRAMERS RULE AND INTERPRETATION:

All newborns should be monitored at subsequent interval of 6-8 hours. Kramer's rule provides mechanism for assessing severity of jaundice clinically. Generally the progression occurs Cephalocaudal to trunk and then to extremities. Keeping these values we can roughly estimate the severity of jaundice. Kramer divided the infants into 5 zones and their SBR values as follows:



Zone	Head and Neck	Chest	Lower body and thigh	Arms and legs	Hands and Feet
SBR mg/dL	5	10	12	15	≥15

These visual inspection can only be used as a guide for evaluation of jaundice. Visual interpretation may always lead to error. Especially newborns with little dark pigmented skin may mislead us. It is just a predictive measure for hyperbilirubinemia.

ASSOCIATED FEATURES IN NEONATAL JAUNDICE:

Blood group refers to the entire blood group system consisting of erythrocyte antigen. They are controlled by series of genes, either allelic or very closely linked genes on a single chromosome.

ABO BLOOD GROUPS:

This is the most significant blood group system in which antibodies are consistently present in serum. They play a vital role in transfusion. These antibodies can cause intravascular hemolysis if incompatible blood is transfused. Normally a reciprocal relationship exist between ABO agglutinogens (antigens) found on surface of RBCs Section and isoagglutinogens (antibodies) found in serum.

People with O blood group will neither have A nor B antigens on RBC and both anti-A and anti-B antibodies were present in serum. A blood group people will have antigen An on RBC and anti-B in serum. B blood group people will have antigen B on RBC and anti-A in serum. AB blood group people will have both antigen A and B on RBC and no antibodies in serum.

Type of blood group	**RBC Antigen**	**Serum Antibodies**	**Occurrences**
A	A	anti-B	35-40%
B	B	anti-A	8-10%
AB	A and B	None	3-5%
O	None	anti-A and anti-B	40-45%

Blood groups are inherited from both the parents (Maternal and Paternal). The ABO blood group system was controlled by a single gene ABO with three types of alleles I^A, I^B and i respectively. The A group is further

divided into 20 sub groups like A1, A2. Cis AB is another rare variant, where A and B genes are transmitted together from a single parent.

Parental Blood Type	A	B	AB	O
A	A or O	A,B,AB or O	A,B or AB	A or O
B	A,B,AB or O	B or O	A,B or AB	B or O
AB	A,B or AB	A,B or AB	A,B or AB	A or B
O	O or A	O or B	A or B	O

ABO incompatibility generally leads to haemolytic diseases in newborns. The breakdown of RBC leads to anemia, jaundice and severe cases it may lead to death.

ABO INCOMPATABILITY IN NEWBORNS:

During pregnancy when the mother and child's blood groups are mismatching, it leads to the breakdown of RBCs of the baby. If the mother is O group, she is prone to this condition. When the baby's blood enters the maternal system its recognized as foreign and mother produces antibodies against it. Mother blood generally do not mix with the babies, both their circulation have been kept separate by placental membrane which serves as a barrier. Occasionally like birth, trauma and miscarriage these two blood gets mixed. When these antibodies pass through the placental membrane it results in breakdown the RBCs of baby. These different blood groups when they come into direct contact the antibodies are formed and leads to the breakdown of RBCs. This increases the production of bilirubin and leads to jaundice. Premature babies are most likely to experience problems because of this ABO incompatibility.

Even after birth these antibodies will persist for weeks in newborns. The cord blood of these babies need to be tested for jaundice. These babies need to be closely monitored to rule out hyperbilirubinemia.

Rh BLOOD GROUPS:

The Rh blood group system is one among the prime important type. Rh antigens are transmembranous proteins with loops exposed at the surface of RBCs. It was named as Rh because it was first discovered form Rhesus monkey. According to the presence or absence it's named as Rh+ and Rh- respectively. These are determined by two alleles at a single locus, they separate

independently of ABO blood group locus. An Rh+ person may be homozygous (+/+) or heterozygous (+/-) and express the D antigen, on the other hand Rh- individual always found to be homozygous (-/-) with absence of D antigen. There are number of Rh antigens like c, C, E and e. These Rh system help to avoid the danger of Rh D incompatibility during pregnancy and transfusion.

Rh+ cells if infused to Rh- individual evoke antibody response (IgG), which is dangerous. During pregnancy Rh incompatibility should be diagnosed. Let us assume a Rh- O group mother carrying an Rh+ A, B or AB foetus. Mother RBCs will develop antibodies against Rh D antigen. In this case the first child may not be affected but subsequent pregnancies these antibodies may cross the placenta and attack the RBCs of RH+ baby. A early pregnancy test for Rh is done and Rh immunoglobulin shot is given to the mother to prevent Rh sensitization.

MINOR TYPES:

INDIAN BLOOD GROUP:

One of the most recently discovered blood group. It is composed of two antigens namely In^a which is of low incidence and In^b of high incidence. These antigens are inherited by co-dominant alleles and denatured by certain enzymes. Anti In^b has been linked with hemolytic transfusion reactions. Anti In^a has not been linked for these reactions. Both antigens has been linked to haemolytic disease of newborns.

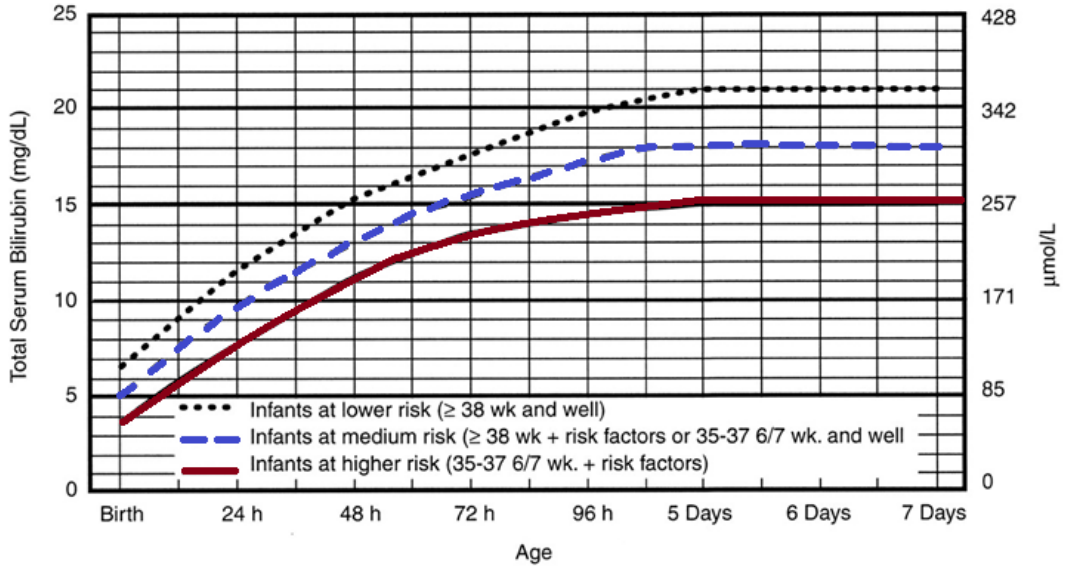
LUTHERAN BLOOD GROUP:

It is also known as Lu blood group. They have Lu^a and Lu^b antigens. Anti Lu^a and Anti Lu^b are uncommon, which are produced after transfusion or pregnancy. They have also been reported only with RBCs stimulation. Mild haemolysis has been reported with these antigens and not much of consideration.

BILIRUBIN VALUES IN NEWBORN

In newborns the normal value of bilirubin depends on birth age i.e., in hours, premature or full term babies. Normal values of bilirubin provided were just for a guideline and they are not same at all places.

PREMATURE BABIES	FULL TERM BABIES
≤ 24 hours of age - below 8.0 mg/dL	≤ 24 hours of age - below 6.0 mg/dL
≤ 48 hours of age - below 12.0 mg/dL	≤ 48 hours of age - below 10.0 mg/dL
3 to 5 days old - below 15.0 mg/dL	3 to 5 days old - below 12.0 mg/dL
≥ 7 days old - below 15.0 mg/dL	≥ 7 days old - below 10.0 mg/dL



- Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin.
- Risk factors = isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis, or albumin < 3.0g/dL (if measured)
- For well infants 35-37 6/7 wk can adjust TSB levels for intervention around the medium risk line. It is an option to intervene at lower TSB levels for infants closer to 35 wks and at higher TSB levels for those closer to 37 6/7 wk.
- It is an option to provide conventional phototherapy in hospital or at home at TSB levels 2-3 mg/dL (35-50mmol/L) below those shown but home phototherapy should not be used in any infant with risk factors.

In full term babies, however if there is a small increase from the above levels doesn't mean that immediate treatment and medical attention to be provided. Where, high levels of bilirubin in any child above normal needs close monitoring and treatment.

HAEMOLYTIC DISEASE OF NEWBORN (HDN)

Early 1600 to 1900 haemolytic disease of newborn has been a mystery, who have been a cause foetal loss temporarily or permanently death. By 1960s trails of therapeutic antibodies given to pregnant women's by United Kingdom and United States prevented HDN. Today we are more concerned about the therapeutic measures and preventing HDN during pregnancy, delivery and further babies development. General cause for haemolytic disease in newborn include ABO incompatibility, Rh hemolysis, G6PD deficiency and other minor blood group incompatibility.

MATERNAL ANTIBODIES AND FETAL RISK:

Incompatibility of Rh blood typing between mother and foetus is major cause of HDN. ABO incompatibility is less severe than Rh with HDN. Hemolysis gets triggered mainly by Rh D antigen, whereas other Rh antigens play a minor role. When an Rh D-ve mother carries a Rh D+ve baby maternal blood is prevented by crossing the barrier placenta. However in first pregnancy only during delivery or caesarean, increased bleeding or complicated labor, there is a mixing of both blood occur. In this situation mother blood produces anti-D against the Rh D antigen. This is retained in her serum as mother gets sensitized to Rh D antigens. During first pregnancy it's not effective much, but during subsequent pregnancies they lead to destruction of fetal RBCs and lead to severe hyperbilirubinemia. If bilirubin level keeps increasing drastically, it may enter the brain and cause kernicterus. A condition that leaves permanent neurological damage if baby survives. Further prolonged destruction of RBCs

Section lead to severe anemia. The liver and spleen have to over produce RBCs to compensate the loss. This leads to hepatosplenomegaly, at times organ dysfunction may also occur. As added complication immature RBCs will circulate in the blood, which will lead to erythroblastosis fetalis. A complication of severe HDN is hydrops fetalis, where the fetal tissues become swollen. This may be fatal to the baby in uterus or after birth.

FIRST PREGNANCY AND SENSITIZATION:

When our immune system first encounters an antigen, its considered to be foreign and produces immune response. This response produces antibody against the antigen, which is called as sensitization.

In case of HDN caused by Rh incompatibility, an Rh D -ve mother encounters D antigen from her Rh D +ve child or a transfusion etc. On first encounter after sensitization the mothers blood produces anti-D. A Direct Coombs test from cord blood has confirmed the presence of anti-D in mother serum. A small amount of fetal blood entry into mother blood is enough for sensitization of mother. This can be brought about by delivery, caesarean, haemorrhage during labor, complicated labor, transfusion, etc. During earlier in pregnancy prenatal bleeding or miscarriage may bring about sensitization. Certain medical procedure like termination of pregnancy may also bring about sensitization. Maternal risk of sensitization is reduced in case of ABO incompatibility. In this case the fetal cells are rapidly destroyed by maternal anti-A or anti-B or both, this prevent the antigen D exposure. Immediate

exchange transfusion followed by phototherapy will prevent the child after birth. If Rh immunoglobulin is given to mother within 72 hours of delivery Rh sensitization can be prevented and we can also protect the next Rh +ve baby.

SUBSEQUENT PREGNANCIES AND HDN:

Initially formed anti-D is of IgM type which cannot cross the placental barrier. In subsequent pregnancies repeated encounter of Rh D antigen produces IgG type anti-D. This can easily cross the placental barrier and enter the fetal circulation. This anti-D attaches to Rh D antigens and thus destroys fetal RBCs. According to hemolysis, HDN may be mild, moderate or high. Mild is within tolerable limits, where at birth mild jaundice or anemia is found and gets resolved with or without treatment.

In case of moderate hemolysis still the level of bilirubin remains low during pregnancy as mother placenta removes it from fetal circulatory path. After birth the immature liver is not able to metabolise the bilirubin. This leads to accumulated increase in bilirubin, which further enters brain and causes kernicterus. Monitoring of mother blood serum levels and ultrasound scan at regular intervals can be preventive.

In case of high levels of prolonged destruction of RBCs lead to sever anemia. Fetal anemia can be corrected by blood transfusion, this replaces the lysed RBCs Section. The liver and spleen increases the production of RBCs to compensate the loss. This leads to hepatosplenomegaly. Immature RBCs enter the circulation and leads to erythroblastosis fetalis. Severe complication of HDN leads to hydrops fetalis, where the fetal tissue swell up.

KERNICTERUS AND HYDROPS FETALIS

KERNICTERUS

Bilirubin a toxic substance to newborns, when it gets elevated in serum they lead to hyperbilirubinemia. Kernicterus is a bilirubin induced neurological problem that affects the brain of newborns. The extra bilirubin found in the circulation gets accumulated as unconjugated bilirubin in the CNS (Central Nervous System) / brain tissues. In grey matter the neurological tissue undergoes heavy destruction by apoptosis and necrosis which provides neurotoxicity. Subsequent damage and scarring of basal ganglia and brain stem nuclei occurs. These extracts of yellow substance has been clearly found in brain, especially grey matter was proven by autopsy of affected newborns. Newborns are mostly affected by hyperbilirubinemia induced kernicterus, which is a potentially fatal condition that leaves permanent neurological damage in surviving babies. This devastating problem to be focused by frequent monitoring of their bilirubin levels, mostly it leads to death.

CLASSIFICATION:

According the characteristic signs and symptom that appear in newborns at various stages they are classified as follows:

I. Acute bilirubin encephalopathy
II. Chronic bilirubin encephalopathy

Acute bilirubin encephalopathy:

The signs and symptoms that appear in early stages is termed as acute bilirubin encephalopathy. This is further observed under 3 stages of newborns as given below

Stage-1

On first few days of life where the babies were observed with extreme jaundice, poor feeding, lethargy and hypotonia.

Stage-2

It occurs on different time and duration where arched back with neck hyper extended backwards (hypertonia), seizures, a high-pitched cry, retrocollis (back ward arching of neck) and opisthotonus (back ward arching of back). Newborns of this stage if not treated properly may lead to future serious illness.

Stage-3

In newborns aged ≥ 1 week where they have low muscle tone (hypotonia)is a typical sign to be monitored. In all these cases the bilirubin content has to be rapidly reduced and brought to normal. If it's not treated properly it leads to chronic bilirubin encephalopathy.

I. Chronic bilirubin encephalopathy:

The signs and symptoms of severe bilirubin induced neurological problems that appear over a prolonged period of time is termed as chronic bilirubin encephalopathy. The chronic onset leads to various clinical manifestations in different stages as follows:

Movement abnormalities include Athetosis (Occurs as a result of basal ganglia), the upper extremities were more affected than the lower ones. Hearing abnormalities include high frequency hearing loss and auditory neuropathy. Visual abnormalities include impaired upper and down ward gaze, strabismus and nystagmus. Dental abnormalities include Hypoplasia and dysplasia. GERD (Gastro esophageal reflux disease) and improper digestion are noted in certain cases. It's prone to mental retardation too.

CAUSES:

The exact role of bilirubin in development of kernicterus can be better understood if the metabolism of bilirubin is clearly understood. Bilirubin is produced when the RBCs are broken down and catabolism of heme occurs. During pregnancy the rate of hemolysis is comparatively higher but still bilirubin level seems to be low because the placenta removes the bilirubin from the fetal circulation. However, after birth drastic increase of bilirubin is noted. The newborn's immature liver is not able to metabolise the increased amount of bilirubin which gets accumulated in the blood. Increased hemolysis and defect in metabolism leads to pile up the accumulation. If the level of bilirubin

continues to increase it may even cross the blood-brain barrier and enter the brain which cause kernicterus.

Newborns cannot completely metabolise the bilirubin for elimination as their liver contains UDPGT (Uridine diPhosphate glucuronosyl transferase) enzyme which is not active for several months. During pregnancy the mother takes up this process where the UDPGT performs a conjugation process called glucuronidation. This process adds large amount of sugar to bilirubin and make it water-soluble. This complex is easier to excrete through urine and faeces. However the serum unconjugated bilirubin level exceeds the binding capacity of albumin, unbound lipid-soluble bilirubin crosses the blood-brain barrier. Albumin bound bilirubin may also cross the blood-brain barrier if damage has occurred because of acidosis, hypoxia, hyperosmolality, asphyxia, or sepsis in newborns. In term newborns with hemolysis, bilirubin levels above 20 mg /dL (342 μ mol / L) is to be taken care as the incidence of kernicterus was found to be associated with these levels and their effects are irreversible.

PREVENTION:

The increased level of bilirubin should be immediately controlled. Screening for hemolysis is highly recommended. If a baby is found with jaundice (increased bilirubin levels) or under the risk factor of jaundice, then phototherapy, intravenous immunoglobulin administration, or exchange transfusion should be recommended. In rare cases combination of Photo

therapy, exchange transfusion and intravenous albumin administration saved few newborns.

TREATMENT:

Various treatment plans for kernicterus is a mystery to solve. However prevention is better than cure. Proper follow-up of newborns and managing the bilirubin levels will avoid kernicterus. Reversal of Kernicterus was comparatively low.

HYDROPS FETALIS

Hydrops refers to severe edematous (swelling) condition in fetus and newborns. When too much of fluid leaves the blood stream and enters the tissues hydrops develop. It's also known as abnormal fluid collection in fetal organ spaces like abdominal cavity, pericardial effusion, pleural effusion, swelling of skin etc. This condition is fatal either intra uterine or immediately after birth.

TYPES OF HYDROPS:

It is further divided into two types:

I. Immune hydrops
II. Non-immune hydrops

I. Immune hydrops:

This occurs because of the blood group incompatibility between mother and baby. Here the mothers immune system causes the breakdown of fetus

RBC's. For example, a mother who has a Rh- Positive who carries Rh- Negative baby, she may develop an immune response that destroys the blood cells of fetus. A greater prolonged destruction of RBCs lead to severe anemia. The liver, spleen, bone-marrow and other organs increase their production of RBCs to compensate the loss. This increased production causes hepatosplenomegaly and they begin to fail. Kidney, adrenal gland and liver dysfunction may also occur. As the heart receives low blood count it may also have to work harder and eventually they also fail. The young blood cells called erythroblast get into the circulation, giving rise to the name as 'erythroblastosis fetalis'(immune hydrops). Rh immunoglobulin a shot given to mother prevent this condition and intra uterine fetal death is controlled.

II. Non-immune hydrops:

It includes all other diseases and complications that interfere with babies concern to manage fluids. Non-immune hydrops is a common type, which is an output of various congenital complication and diseases. Severe anemia, congenital infections (syphilis, parvovirus), liver diseases, spleen diseases, chromosomal abnormalities and birth defects (cystic adenomatoid malformation, polycystic kidneys) were causes of non-immune hydrops.

SYMPTOMS:

The following are the most common symptoms found in hydrops fetalis:

During pregnancy collection of large amounts of amniotic fluid (polyhydramnios), thickened placenta, enlarged (heart, liver, spleen), under developed lungs, fluid accumulation around abdomen, heart and lungs have been found by ultrasound.

After birth the newborn was witnessed with pale colouration, severe edema localized or generalized, enlarged (liver, spleen), severe respiratory distress, hypoglycaemia, generally swollen and have round abdomen.

PREVENTION:

Generally complete family history was worked out as background information. Patients medical, social history and physical examination were clearly worked out. As a part of diagnostic procedure ultrasound, amniocentesis, fetal blood sampling, fetal echocardiogram, prenatal ultrasonography, blood test of mother for incompatibility helps greatly to prevent hydrops fetalis.

TREATMENT:

When the cause for hydrops fetalis is found, like gestational age, extent of disease, babies condition and tolerable limits the treatment is planned accordingly. If the newborn is found with potentially low blood count, then transfusion of blood will be effective. In case of excessive fluid collection around lung and abdomen can be removed by a needle. Gene therapy may provide therapeutic promise for future.

METHODS OF DIAGNOSIS

KRAMERS METHOD:

Direct visual examination of jaundice is not a good measure of bilirubin level in blood. Here the entire fetal body is divided into 5 zones and their corresponding SBR provide. This can be used as a guide and not for accuracy.

TOTAL SERUM BILIRUBIN:

Total serum bilirubin can be measured both directly and indirectly. TSB level was a good standard examination. However these facilities were available in research facilities only. To be precise there were no standard levels to predict what level sets a guideline for jaundice. We generally have to go by with average values. Only by post mortem evidences it's found that babies with lower SBR levels are at risk of kernicterus.

TRANSCUTANEOUS BILIRUBIN METER: (TCB)

Transcutaneous bilirubin levels can be measured with a hand held device TCB (Transcutaneous bilirubinometer), which measures bilirubin levels when slightly pressed against skin surface. It is just a screening test.

ADVANTAGE:

One advantage of this method is no need to puncture skin. This technique is based on multiple wavelength analysis. Measures serum bilirubin independent of skin pigmentation, postnatal age and weight.

DISADVANTAGE:

It provides an underestimate of TSB. Still the result were not to satisfactory levels. Unreliable after phototherapy due to breaching of skin.

PRECAUTION:

Check the TSB if TCB ≥ 70th percentile of TSB value for phototherapy. TCB ≥ 75th percentile. TCB ≥ 13 mg/dL at follow up or after discharge.

END TIDAL CARBON MONOXIDE: (ETCoc)

It gives the factors that contribute to neonatal jaundice (Haemolysis/Conjugation defects)

Blood grouping and Rh typing of mother and baby.

Peripheral smear study with reticulocyte count.

HAEMATOCRIT VALUE:

Generally detects polycythemia. It also suggest blood loss from occult haemorrhage.

DIRECT COOMBS TEST:

Identification of antibodies on infant RBCs.

G6PD SCREENING:

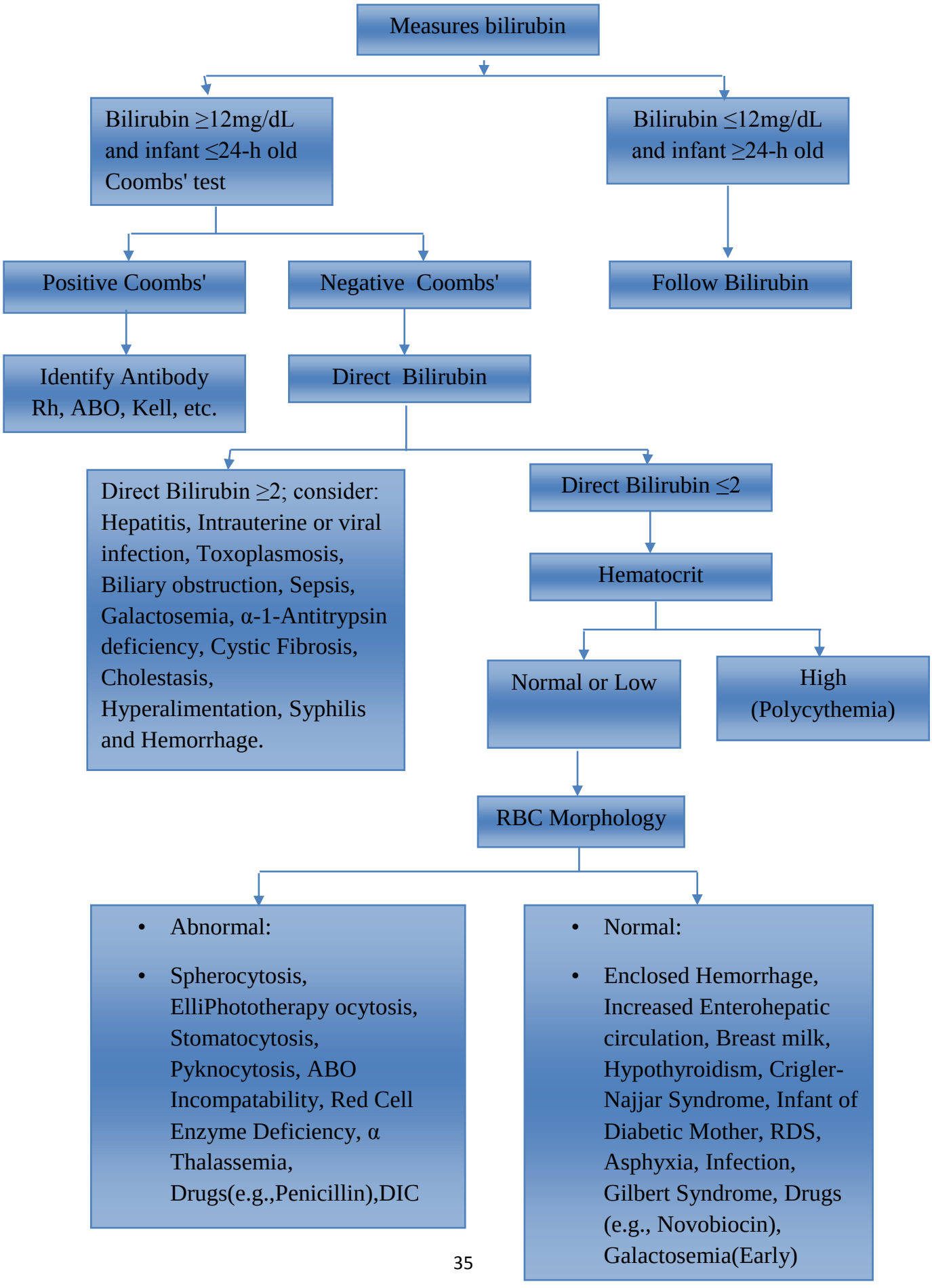
This may be helpful especially in male infants. G6PD deficiency is more common among African American males. Screening parents for G6PD

deficiency is helpful in making diagnosis of hyperbilirubinemia. Infants who had G6PD deficiency and discharged early have been reported with severe hyperbilirubinemia and sequelae.

HEEL STICK TEST:

In this technique blood sample from heel of the baby is collected. A sterile lancet is used to puncture the heel, under aseptic condition the blood sample derived from baby and stored for analytical purposes.

Approach to Clinical Jaundice



PREVENTION AND TREATMENT

PHOTOTHERAPY

In phototherapy the baby lies in a incubator (enclosed plastic crib). The light emitted with specific wavelength, intensity and proper skin exposure makes the bilirubin water soluble. Bilirubin absorbs visible light with wavelength ranging between 400-500nm. Special blue lamps with peak output of 425-475nm is found to be effective. These water soluble bilirubin can be easily eliminated with excretion. Phototherapy can be used to prevent hyperbilirubinemia with increased TSB levels in newborns as well initial therapy in case of severe hyperbilirubinemia.

TECHNIQUE OF PHOTOCHEMICAL REACTION:

Generally it occurs in extra vascular space of skin. The process of isomerization changes bilirubin into water soluble isomers, that's passed out without getting stuck in liver. The natural isomer of UCB (4Z, 15Z) is converted to 4Z, 15E polar isomer that gets diffused into circulation and excreted into the bile. If the excretion is slow the photo isomer is converted back to UCB and reabsorbed from the gut. Even at cases bilirubin level remains same, their toxicity has been reduced by presence of these photo isomers about 20% at equilibrium.

Structural isomerization consist of intramolecular crystallization of bilirubin, which results in the formation of lumirubin. Lumirubin constitutes 2

to 6 % of TSB concentration. Conversion of bilirubin to lumirubin is irreversible. It is excreted through bile and urine. Lumirubin is responsible for therapeutic effect of phototherapy in lowering bilirubin levels.

INDICATIONS OF PHOTOTHERAPY:

The phototherapy technique is used when bilirubin level increases and proves fatal to infants. During last few years, autopsy has proved that immature infants were at risk of bilirubin encephalopathy at lower TSB levels than mature infants. Newborns with haemolytic disease and increased serum bilirubin levels were subjected to phototherapy. On special occasion like ELBW infants, severely bruised infants and slight increase in TSB levels were immediately provided with phototherapy as prophylactic measure. Test for bilirubin-albumin binding or unbound bilirubin ration is used in certain cases. If the indirect bilirubin levels were not high, recommendation of phototherapy may lead to "Bronze Baby" syndrome. Exchange transfusion more safer than phototherapy.

TYPES OF PHOTOTHERAPY:

Newborns with jaundice were treated with various light spectrum called phototherapy. Various types of phototherapy as follows:

BLUE LIGHT:

Special blue lamps with wave length output 425-475 nm, is most effective in treatment of neonatal jaundice. Generally newborns with TSB levels greater than 20mg/dL were treated with this blue light. The results were observed every 2, 4, 6, 8 12, 24, 36 and 48 hours. Final result proved satisfactory that bilirubin levels get reduced.

WHITE LIGHT:

Generally white lights are less effective compared to others. Their wave length varies from 380-700nm. Decreasing the distance between lamp and the baby increases effectiveness.

GREEN LIGHT:

This type of light is not in common use because the time and duration of exposure is comparatively longer to have significant results.

LIGHT BANKS:

It consist of alternating special blue and day light. Fluorescent lights are more effective compared to normal ones.

SPOT PHOTOTHERAPY:

Used in newborns under radiant warmers. Here the baby is place on fiberoptic blanket overhead with quartz halide white light with blue spectrum output.

DOUBLE PHOTOTHERAPY:

In this blue green spectrum of light has been used which proves to be more useful. The baby lies on fiberoptic blanket, where the phototherapy lights were found to be overhead. Concurrent use of two or more phototherapy units for same baby is made possible. Newborns with high TSB levels use this type.

DOS AND DONTS:

Newborn are kept naked to ensure that full body is exposed to phototherapy. Eye padding should be done to avoid retinal damage. Distance between the newborn and the baby should be less than 20 inches. Newborn should be placed at the centre of the circle of light, to ensure full effect of phototherapy. Basic fluid requirement should be met by frequent feeding or it may lead to dehydration. Phototherapy is discontinued when the TSB level falls less than 1.5-3mg/dL of the initial value.

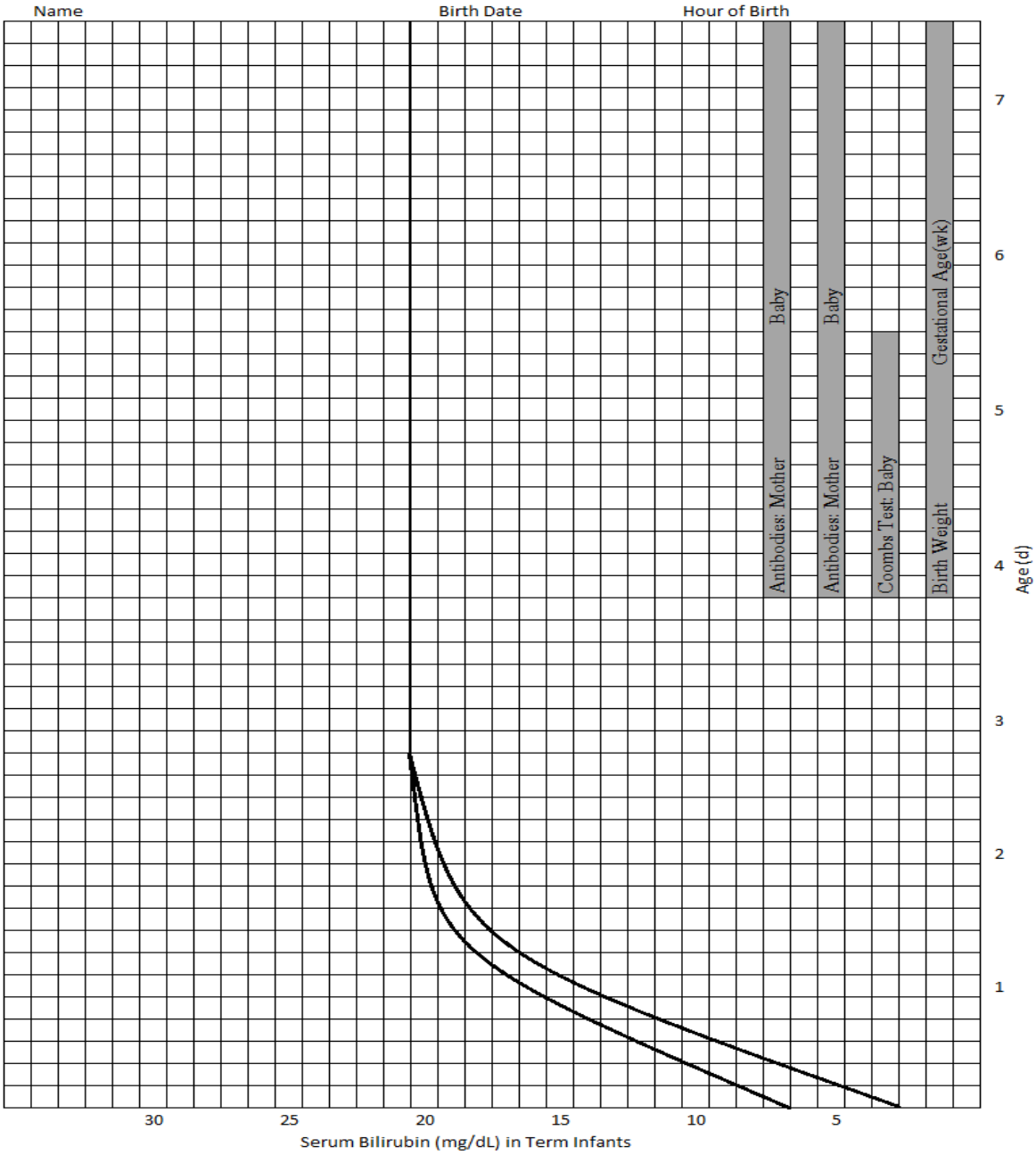
SIDE EFFECTS OF PHOTOTHERAPY:

Various side effects include skin rash, insensible water loss, retinal damage, dehydration or diarrhoea, difficulty in maintaining Homeostasis (Hypothermia/Hyperthermia), redistribution of blood flow leading to PDA (Patent Ductus Arteriosis) in preterm newborn, hypocalcemia, tanning of skin/ Bronze baby syndrome, sister chromatids exchanges or breaking of DNA strand occurs and reduction of tryptophan in aminoacid solution.

EXCHANGE TRANSFUSION (ET)

When phototherapy fails to control bilirubin neurotoxicity, exchange transfusion stands as second life saving treatment. It is a process of removing haemolysed and antibody coated erythrocytes and replacing it with donor blood. This removes blood toxins and certain blood components, as well replacing it with adequate blood. As a prophylactic treatment early exchange transfusion was performed which avoids fatal results.

Generally early exchange transfusion is done in anemia with cord haemoglobin $\leq$ 11gm/dL, elevated cord bilirubin $\geq$ 4.5mg/dL or both. Severe increase in TSB level more than 1mg/dL/hr and $\geq$ 0.5mg/dL/hr with moderate anemia, when bilirubin level $\geq$ 20 mg/dL or if there is progression of anemia inspite of controlled bilirubin values is an indication for exchange transfusion.



Serum Bilirubin levels plotted against age in term infants with erythroblastosis. Infants with levels plotting below the bottom line require no action, those with levels between two lines should receive phototherapy, and those with levels above the top line should undergo exchange transfusion.

Blood volume used in exchange transfusion for term infants 80ml/kg (1 x circulating blood volume), is called single volume transfusion. Blood volume used in exchange transfusion for newborn infants 2 x 80 ml/kg is called double volume transfusion. This replaces about 87% of newborns blood with new blood.

BLOOD USED FOR TRANSFUSION:

Fresh whole blood (≤ 7 days) irradiated and FFP collected in CPD is used. In Rh incompatibility O -ve cross matched blood against mother is used. In ABO incompatibility O -ve or Rh compatible and cross matched with mother and infant is used. Blood should not contain sensitizing antigen and cross matched with mother for other isoimmune hemolytic disease. In non immune disease blood is typed and cross matched against newborn.

TECHNIQUE OF EXCHANGE TRANSFUSION:

Done in newborn placed under warmer with vital monitoring. Newborns extremities has to be restrained carefully. Appropriate blood volume should be calculated for newborn before transfusion. Blood should be pre warmed to 37 degree Celsius. Strict aseptic condition is maintained umbilical vein line is secured. Do exchange transfusion by pull push technique in aliquots of 5ml for ≤ 1.5 kg, 10ml for ≥ 1.5 -2.5 kg, 15ml for ≥ 2.5-3.5 kg and 20ml for ≥ 3.5kg was recommended. General time period recommended for exchange transfusion is 1 hour. Every 4 hours of interval SBR levels to be checked after transfusion and phototherapy is continued.

COMPLICATIONS OF PHOTOTHERAPY:

Metabolic complication include Hypocalcemia, hypomagnesaemia, hypoglycaemia and hyperkalemia. Acid base imbalance includes late metabolic alkalosis (If the baby has healthy liver), acidosis (If the baby is sick and not able to metabolise citrate). Iatrogenic causes are vessel perforation, embolization,

thrombosis, infarction, arrthymia and volume over load. Thrombocytopenia and decreased clotting factor induced bleeding. Blood born infections are sepsis, AIDS, CMV, HIV and malaria. Graft Versus host disease are prevented by using irradiated blood.

RECENT MODALITIES:

IVIG :

In Rh and ABO incompatibility administration IVIG is found to significantly reduce the need for exchange transfusion. Recommended dose varies from 500-1000mg/kg. This can be used in combination with intensive phototherapy. IVIG was not much useful if the newborn was anaemic (Hb ≤ 10mg/kg)

ORAL PHENOBARBITONE:

It reduces the bilirubin content of blood by inducing microsomal enzymes increases bilirubin conjugation and excretion.

ORAL AGAR:

It's given to the newborns with bilirubin values ≥ 15 mg/dL (breast and formula fed newborns) to reduce the bilirubin level in blood. Generally the mechanism of action is by decreasing enterohepatic circulation.

METALLOPROTO PORPHYRINS:

Tin and zinc were most commonly used. Mechanism of action is by competitive inhibition of heme oxygenase. They are used to treat Coombs +ve ABO incompatibility and Crigler Najjar type-1 patient with hyperbilirubinemia.

AIMS AND OBJECTIVE

To evaluate the predictive value of umbilical cord bilirubin in identifying term newborns with ABO /Rh incompatibility for subsequent hyperbilirubinemia in 1st week of life.

METHOD OF STUDY:

Prospective clinical study in Tiruneveli Medical College Hospital (TVMCH) which is carried out in all consecutive term newborns with ABO /Rh incompatibility over a period of 8 months (February 2015 to September 2015) duration.

The study population was initially followed up clinically by Kramer's method and by transcutaneous bilirubinometer. Newborns identified with jaundice were followed up using serial serum bilirubin values.

METHOD OF ASSAY - SERUM BILIRUBIN:

Total serum bilirubin, conjugated bilirubin, unconjugated bilirubin were obtained via the calorimetric diazo method.

3ml of cord venous blood was taken in a sterile syringe, put in clean capped tube and sent immediately to the hospital lab. Serum was used for analysis most importantly samples were protected from light during processing and storage. The haemolysed samples were then excluded.

INCLUSION CRITERIA:

All consecutively born babies in Tirunelveli Medical College Hospital irrespective to mode of delivery and gender were included. Term newborns with gestational age of 36-40 weeks were included. APGAR score of over 7 at the first minute and 10 at fifth minute of life is taken. Absence of significant illness or major congenital malformation.

EXCLUSION CRITERIA:

Term newborns with significant illness like sepsis, respiratory distress syndrome, infant of diabetic mother, asphyxia that could aggravate hyperbilirubinemia. Low birth weight newborns (≤ 2kg).

REVIEW OF LITERATURE

MEASUREMENT OF BILIRUBIN:

There is no difference between capillary and venous blood sampling for bilirubin. Bhutani et al used capillary and venous sampling in his studies. TcB (Transcutaneous bilirubinometer) is unreliable after phototherapy or notifiable colour changes in skin and its thickness. It can be only used as screening tool or in predicting low levels of serum bilirubin.

TCB AND TSB:

Maisels et al used strategies for determining bilirubin when TSB measurement is indicated. Measurement of TSB should be performed on following conditions;

1. TCB value ≥ 70% of TSB level

2. TCB more than 75th percentile on Bhutani namogram

3. TCB value ≥ 30 mg/dL at follow up or discharge

ASSOCIATION WITH RATE OF CONFIGURATIONAL ISOMERISATION IN PHOTOTHERAPY WITH HB LEVEL:

In phototherapy, formation of photoisomers i.e., Z to E form is significant after only 15mts. Mreihil K et al, has found that initial rate of isomerization is indirectly proportional to the HB level.

ASSOCIATION OF PHOTOTHERAPY AND HIGH LEVEL OF SERUM BILIRUBIN:

Adelia and Cancciacao 2004 studies state that phototherapy was significantly associated with high levels of CORD BILIRUBIN values i.e., 2.1 ± 0.46 mg/dL and level with no phototherapy is 1.75 ± 0.46 mg/dL.

LEVEL OF SERUMBILIRUBIN AND JAUNDICE:

Alpay et al, in his study used serum bilirubin level of 120 μ mol/L in predicting development of neonatal jaundice and levels that has been less than 120 μ mol/L in first day didn't develop any symptoms related to hyperbilirubinemia. This study was supported by Stevenson et al.

PRETERM NEONATES WITH JAUNDICE AND VITAMIN SUPPLEMENTATION:

Ballin et al, states that high vitamin C supplementation leads to heins body hemolytic anemia in preterm infants. Doyle et al, work didn't support the above study. Bass et al, state that administration of vitamin C is safe but not associated with hemolysis. Ojo et al, stated decreased vitamin E in term

newborn is associated with increase in bilirubin level and development of jaundice.

ASSOCIATION OF CORD BILIRUBIN AND GENDER:

In a specific study correlation exist between levels of cord bilirubin and jaundice development in 1st week of life. Study population comprises of 50 males and 44 females which includes both term and preterm. In preterm cord bilirubin is found to be higher in male gender. In term babies cord bilirubin is equal in both genders i.e., there was no satisfactory significant difference between them.

The above study matched with Amar et al, Rostami and Mehrabi. This study was disagreed with Rudy et al.

ASSOCIATION OF CORD BILIRUBIN WITH BIRTH WEIGHT AND GESTATIONAL AGE:

Matthias et al in his study concluded that CORD BILIRUBIN is negatively correlated with gestational. Adelia and Canceicao in his study proved that there was no correlation between CORD BILIRUBIN and Birth weight.

ASSOCIATION OF CORD BILIRUBIN WITH MODE OF DELIVERY:

Rostami, Mehrabi and Amar et al stated there was no significant difference between normal delivery and caesarean section.

ASSOCIATION OF CORD BILIRUBIN WITH BLOOD GROUPS:

In one study ABO incompatibility was found in 13.8% of all study group and 3.2% only with Rh incompatibility. This indicates the mean CORD BILIRUBIN value is significant among ABO incompatibility compared to negative ones in both groups.

Adalia and Canceicao stated that no significant difference in CORD BILIRUBIN between blood group incompatibility.

CUT OFF POINTS OF CORD BILIRUBIN IN TERM AND PRETERM:

Rudy et al used ROC analysis and states that CORD BILIRUBIN more than 2.5 mg/dL had high specificity and sensitivity. Amar et al found in his studies that CORD BILIRUBIN more than 2 mg/dL had high sensitivity and this critical bilirubin level had very high negative predictive value and fairly low positive predictive value.

Rostami and Meharabi in their study figured out that CORD BILIRUBIN more than 3 mg/dL is not a useful predictor of jaundice. Suresh and Clark stated that CORD BILIRUBIN could predict the development of jaundice in term newborn. Rataj et al from his studies concluded CORD BILIRUBIN $\geq$ 2.5 mg/dL had probability of 89% possibility of development of jaundice.

STATISTICAL ANALYSIS

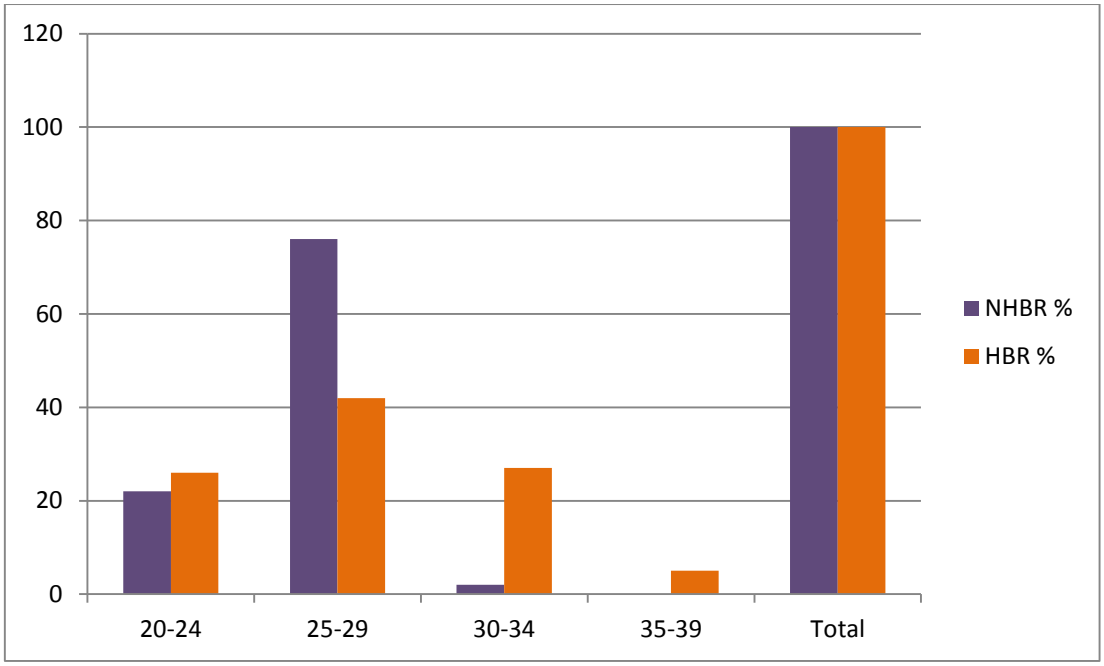
During the first week of neonatal, the incidence of Hyperbilirubinemia was classified as positive and the others were treated as negative. The measurable variables were analyzed and interpreted between them by the student's t test and the ordinal and categorical variables between them were interpreted by Chi- square (χ^2) test. The risk factors were interpreted by binary logistic regression. The predictive value of incidence was estimated by the ROC curve. The statistical procedures were performed with the help of an appropriate statistical package or manually. The P-values less than 0.05 ($P<0.05$) was considered as statistically significant.

RESULTS:

The study subjects of 150 newborn babies were observed for 1week and those who have developed Hyperbilirubinemia and they were classified as HYPERBILIRUBINEMIA and the remaining babies were treated as ordinary babies. The various analysis and interpretations were made by comparing the two groups. The groups were described according to their Mothers age, Gestational age, parity mode of delivery mother's blood group and the neonate's blood group.

Table:1. Age of mothers compared between the HYPERBILIRUBINEMIA and NONHYPERBILIRUBINEMIA:

Mothers' age group	NONHYPERBILIRUBINEMIA		HYPERBILIRUBINEMIA		Total	
	Frequency	%	Frequency	%	Frequency	%
20-24	11	22.0	26	26	37	24.7
25-29	38	76.0	42	42	80	53.3
30-34	1	2.0	27	27	28	18.7
35-39	0	0.0	5	05	5	3.3
Total	50	100.0	100	100.0	150	100.0
Mean ±SD	25.9±2.1		27.2±4.1			
Significance	P<0.05					

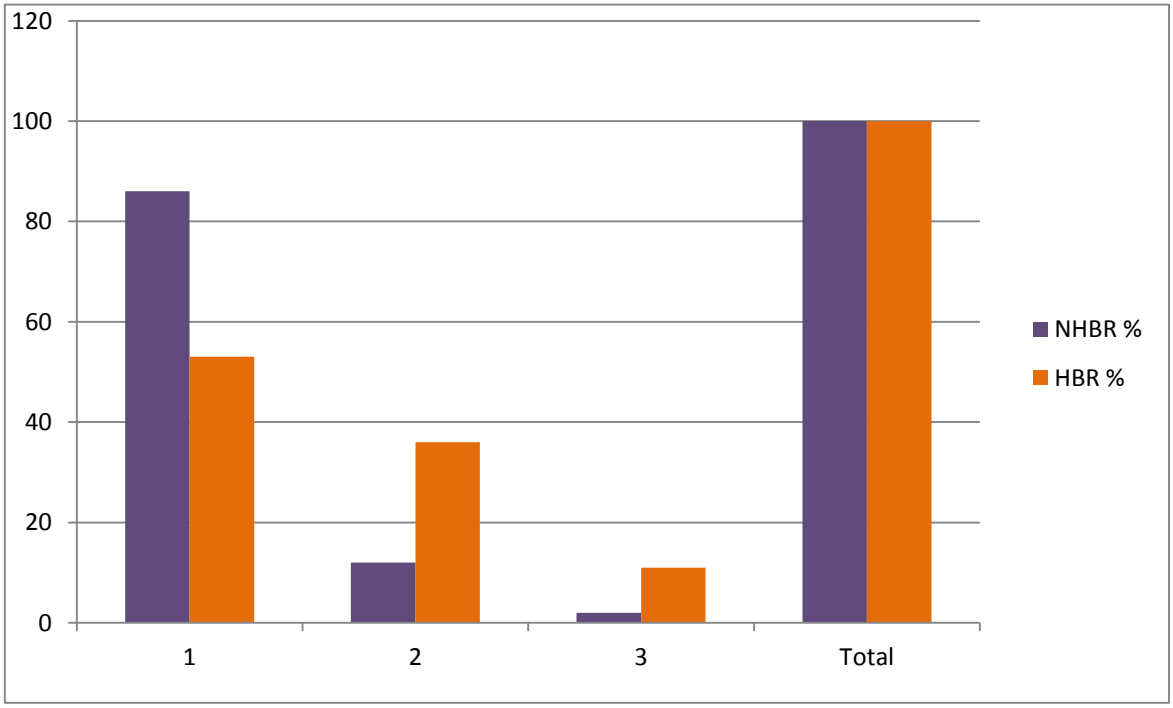


In the above the mean age NONHYPERBILIRUBINEMIA(Non Hyperbilirubinemia) neonatal Mothers was 25.9±2.1 years. The mean age of HYPERBILIRUBINEMIA(Hyperbilirubinemia) was 27.2±4.1 years. The difference between the above means was statistically significant, ($P<0.05$).

RELATIONSHIPS OF MATERNAL AND PERINATAL CHARACTERISTICS WITH NONHYPERBILIRUBINEMIA AND HYPERBILIRUBINEMIA:

Table-2: The association between the parity with the NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA:

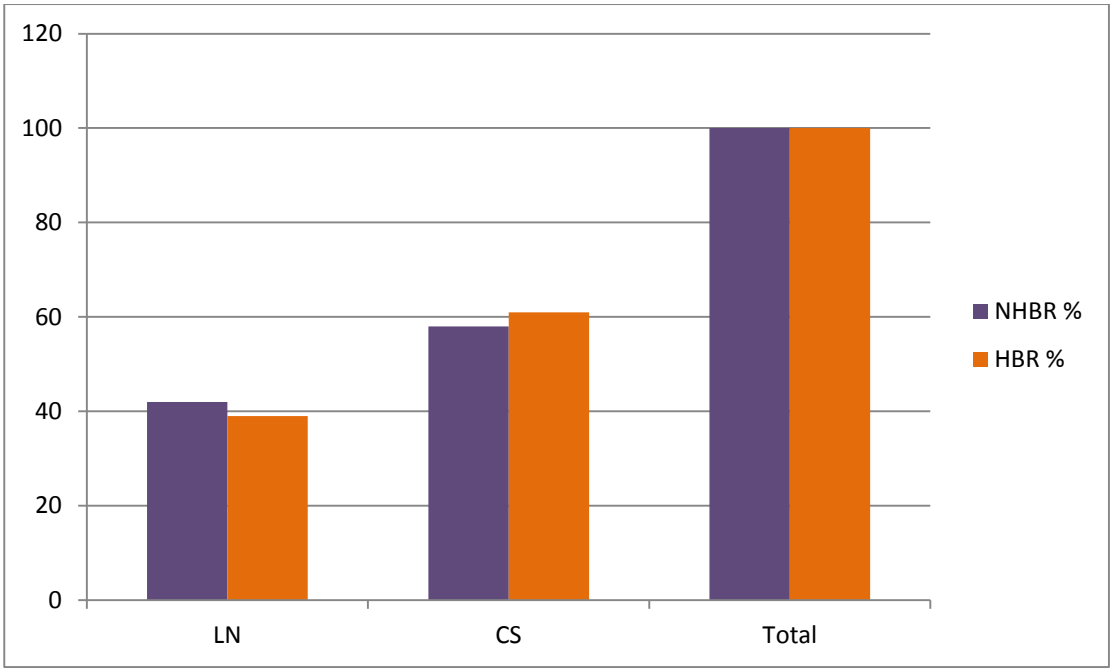
Parity	NONHYPERBILIRUBINEMIA		HYPERBILIRUBINEMIA		Total		χ^2	df	significance
	No	%	No	%	No	%			
1	43	86.0	53	53.0	96	64	15.904	2	P<0.001
2	6	12.0	36	36.0	42	28			
3	1	2.0	11	11.0	12	8.0			
Total	50	100.0	100	100.0	150	100.0			



In the above table the association between the parity of mother with the babies of NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA. The results revealed that the primy para neonates were associated with NONHYPERBILIRUBINEMIA and the multi para neonates were associated with HYPERBILIRUBINEMIA. The relationship was statistically very highly significant ($p<0.001$).

Table-3: Mode of deliveries and association with NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA:

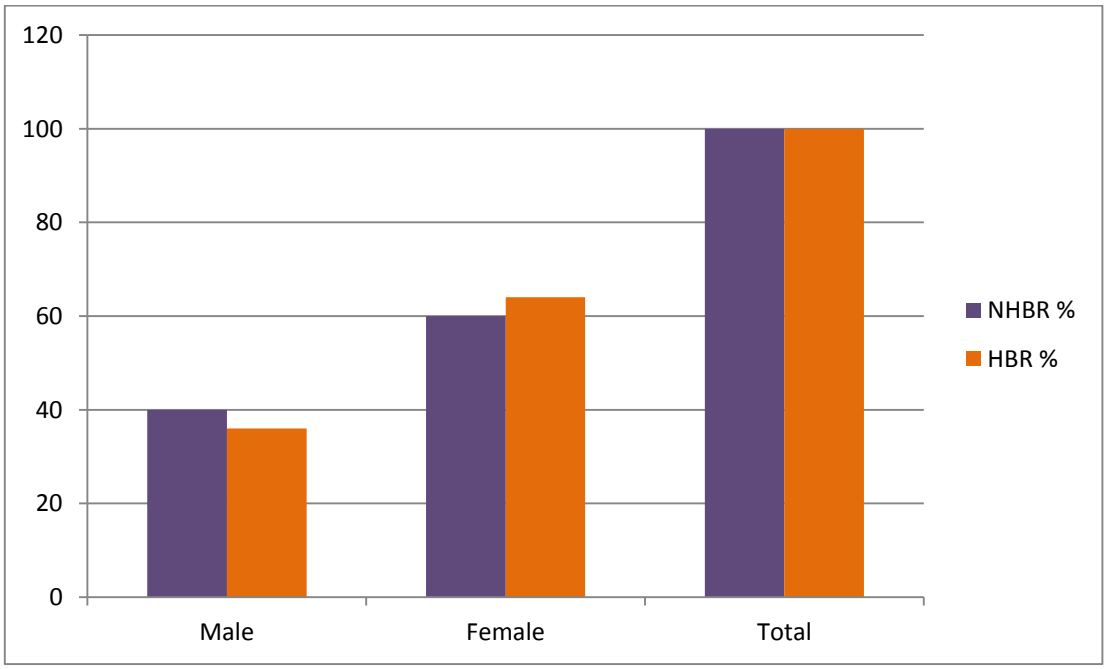
Mode of Delivery	NONHYPERBILIRUBINEMIA		HYPERBILIRUBINEMIA		Total		χ^2	df	significance
	No	%	No	%	No	%			
LABOUR NATURAL	21	42.0	39	39.0	60	40.0	0.125	1	P>0.05
CAESAREAN SECTION	29	58.0	61	61.0	90	60.0			
Total	50	100.0	100	100.0	150	100.0			



The table -3 Shows that there was no statistically significant association (P>0.05).

Table-4: The association between the gender of neonates with NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA:

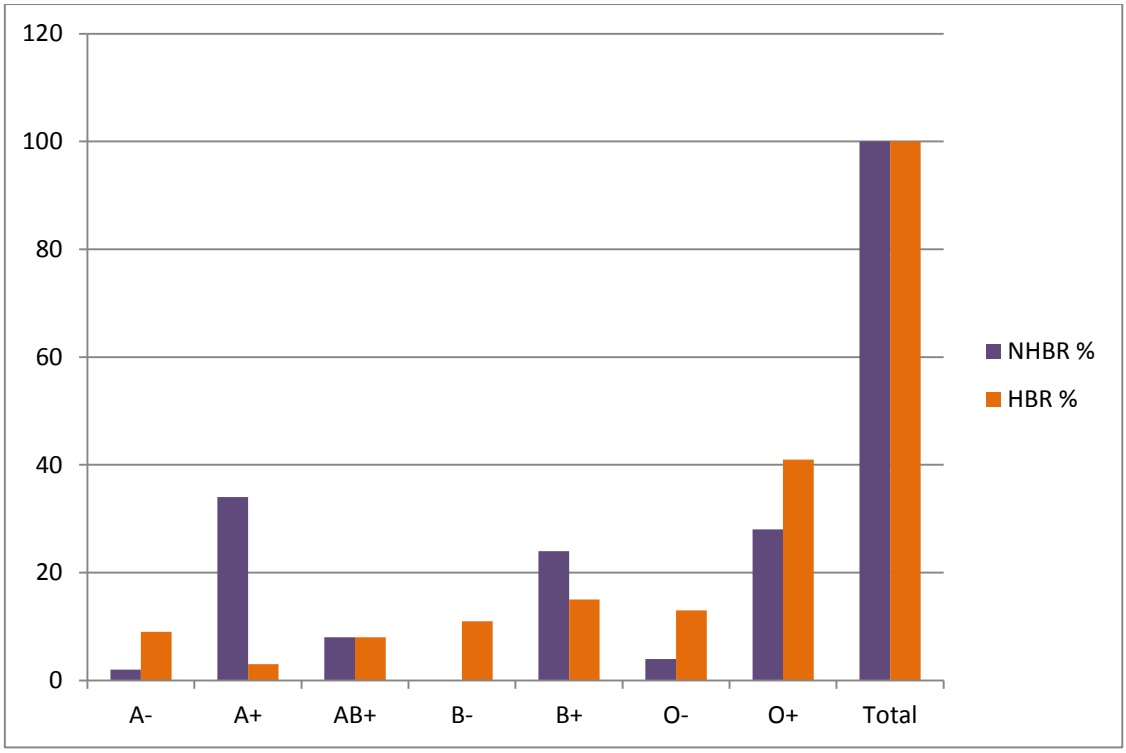
Gender	NONHYPERBILIRUBINEMIA		HYPERBILIRUBINEMIA		Total		χ^2	df	significance
	No	%	No	%	No	%			
Male	20	40.0	36	36.0	56	37.3	0.228	1	P>0.05
Female	30	60.0	64	64.0	94	62.7			
Total	50	100.0	100	100.0	150	100.0			



The NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA did not have any significant association with the gender of the baby (P>0.05).

Table-5. Mothers' blood group associated with incidence of HYPERBILIRUBINEMIA and NON**HYPERBILIRUBINEMIA:**

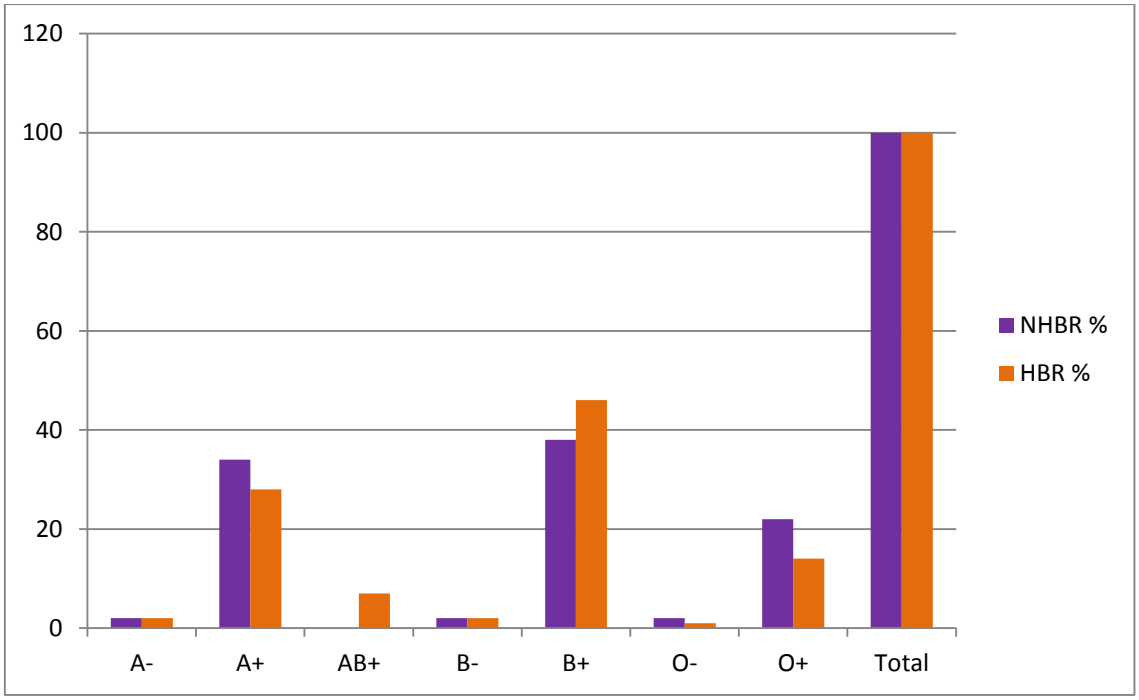
Blood group	NONHYPERBILIRUBINEMIA		HYPERBILIRUBINEMIA		Total		χ^2	df	Significant
	No	%	No	%	No	%			
A-	1	2.0	9	9.0	10	6.7	37.711	6	P <0.001
A+	17	34.0	3	3.0	20	13.3			
AB+	4	8.0	8	8.0	12	8.0			
B-	0	0.0	11	11.0	11	7.3			
B+	12	24.0	15	15.0	27	18.0			
O-	2	4.0	13	13.0	15	10.0			
O+	14	28.0	41	41.0	55	36.7			
Total	50	100.0	100	100.0	150	100.0			



The Mothers' blood groups A-, B-, O- and O+ were associated with HYPERBILIRUBINEMIA neonates and A+ and B+ were associated with NONHYPERBILIRUBINEMIA. The associations were statistically very highly significant ($P<0.001$).

Table-6. Neonates' blood group associated with incidence of HYPERBILIRUBINEMIA and NONHYPERBILIRUBINEMIA:

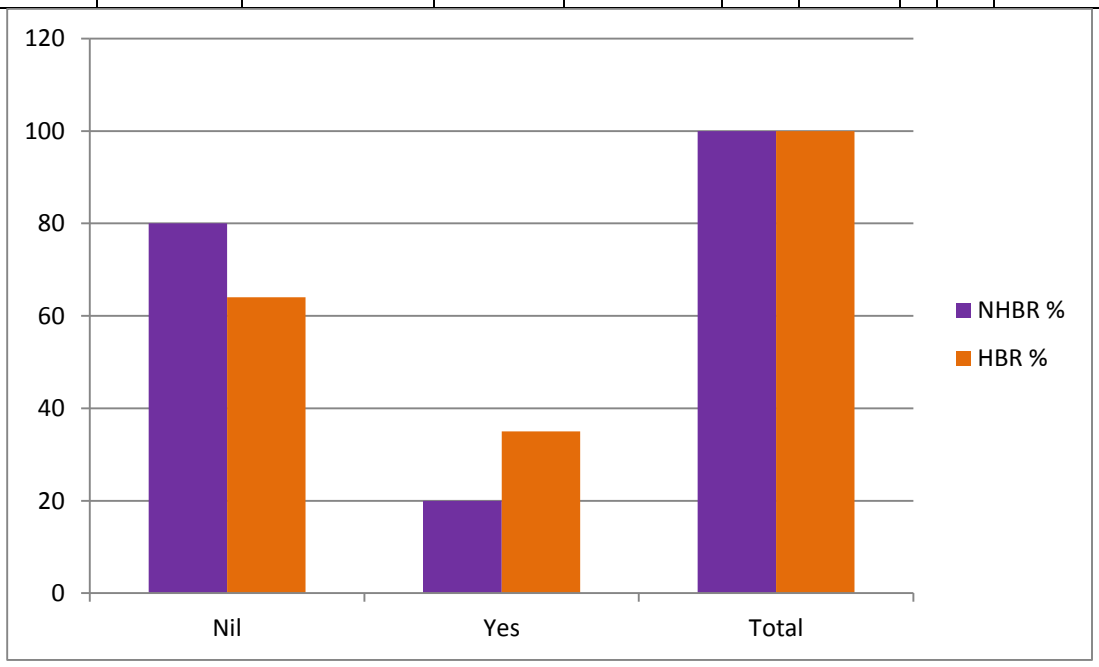
Blood group	NONHYPERBILIRUBINEMIA		HYPERBILIRUBINEMIA		Total		χ^2	df	Significant
	No	**%**	**No**	**%**	**No**	**%**			
A-	1	2.0	2	2.0	3	2.0	5.922	6	P>0.05
A+	17	34.0	28	28.0	45	30.0			
AB+	0	0.0	7	7.0	7	4.7			
B-	1	2.0	2	2.0	2	1.3			
B+	19	38.0	46	46.0	46	30.7			
O-	1	2.0	1	1.0	1	0.7			
O+	11	22.0	14	14.0	14	9.3			
Total	50	100.0	100	100.0	150	100.0			



The neonates blood groups were not significantly associated with NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA ($P>0.05$).

Table-7: The association between the ABO incompatibility with NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA:

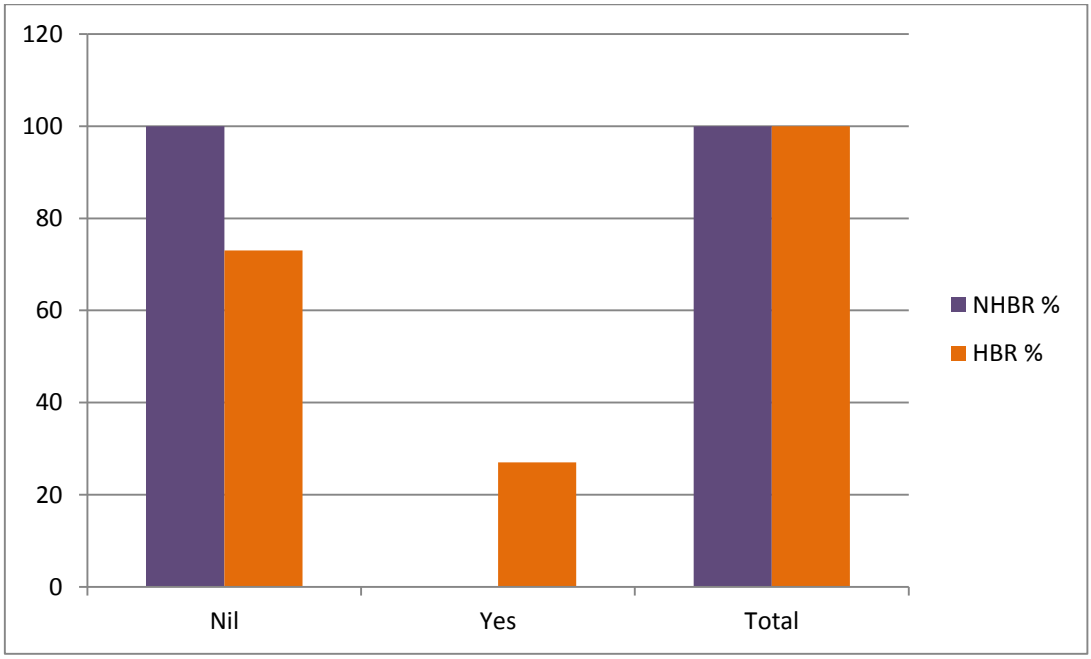
ABO incompat.	NONHYPERBILIRUBINEMIA		HYPERBILIRUBINEMIA		Total		χ^2	df	significance
	No	%	No	%	No	%			
Nil	40	80.0	64	64.0	104	69.3	4.013	1	P <0.05
Yes	10	20.0	36	35.0	46	30.7			
Total	50	100.0	100	100.0	150	100.0			



The ABO incompatibility shown in the above table had associated with the HYPERBILIRUBINEMIA (35%) and nil associated with NONHYPERBILIRUBINEMIA (80%). These associations were statistically significant (P<0.05).

Table-8: The association between the Rh incompatibility with NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA:

Rh incompat.	NONHYPERBILIRUBINEMIA		HYPERBILIRUBINEMIA		Total		χ^2	df	significance
	No	%	No	%	No	%			
Nil	50	100.0	73	73.0	123	82.0	14.685	1	P<0.001
Yes	0	0.0	27	27.0	27	18.0			
Total	50	100.0	100	100.0	150	100.0			



The Rh incompatibility shown in the above table had associated with the HYPERBILIRUBINEMIA (27%) and nil associated with

NONHYPERBILIRUBINEMIA (100%). These associations were statistically very highly significant ($P<0.05$).

COMPARISON OF PERINATAL CHARACTERISTICS NBHR AND HYPERBILIRUBINEMIA:

The perinatal characteristics of neonatal such as gestational age, birth weight, Hb, Apgar scores 1M and 5M were compared between NHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA.

Table:9. Comparison of perinatal characteristics between NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA:

Variable	NONHYPERBILIRUBINEMIA, n=50		HYPERBILIRUBINEMIA, n=100		Difference b/w means	't'	df	Significance
	Mean	SD	Mean	SD				
GA	36.5	0.5	36.6	0.5	0.1	1.874	148	P>0.05
BW	2.6	0.3	2.7	0.3	0.1	0.278	148	P>0.05
HB	14.2	2.1	15.6	0.7	1.4	5.928	148	P<0.001
Apcar1m	7.2	0.4	7.1	0.3	0.1	1.527	148	P>0.05
Apcar5 m	8.16	0.4	8.18	0.4	0.02	0.303	148	P>0.05

In the above variables except HB, the others did not differed significantly. The mean HB of NONHYPERBILIRUBINEMIA was 14.2±2.1gms/dl and the same of HYPERBILIRUBINEMIA was 15.6 ±0.7gms/dl. The difference between them was statistically very highly significant (P<0.001).

CORD BILIRUBIN CHARACTERISTICS:

Table:10. Comparison of total CORD BILIRUBIN and SBR between NON-HYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA at birth:

Total	NONHYPERBILIRUBINEMIA, n=50		HYPERBILIRUBINEMIA, n=100		Difference b/w means	't'	df	Significance
	Mean	SD	Mean	SD				
CORD BILIRUBIN	2.3	0.3	4.0	0.5	1.7	21.357	148	P<0.001
SBR	2.6	0.4	8.8	2.6	6.2	16.486	148	P<0.001

The mean CORD BILIRUBIN total of two groups was 2.3±0.3 and 4.0 ± 0.5. The difference was statistically significant (P<0.001). Similarly the mean difference between them was statistically very highly significant (P<0.001).

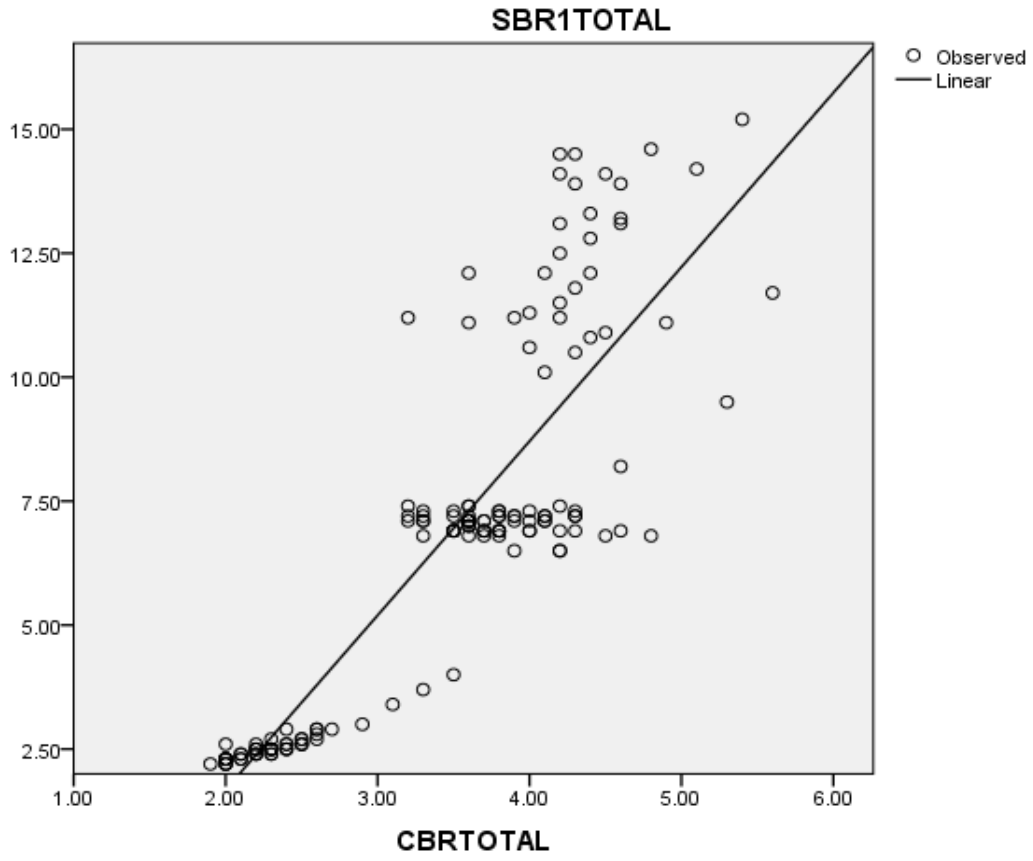
Table-11: Relationship between total CORD BILIRUBIN and SBR:

Variables	n	r	significance	r^2	% of r^2
Total CORD BILIRUBINXSBR	150	0.861	P<0.001	0.741	74.1

The relationship between Total CORD BILIRUBIN and SBR was statistically very highly significant (P<0.001).

Regression equation for estimating the total SBR through total CORD BILIRUBIN:

Fig-1:Regression Curve of relation between CORD BILIRUBIN total with SBR total.



Regression equation: SBR (T)= 3.513CORD BILIRUBIN(T) – 5.343.

PREDICTION OF CORD BILIRUBIN (T) CUT OFF VALUE:

The variables such as CORD BILIRUBIN total, gestational age, gender, ABO incompatibility and Rh incompatibility were inter related variables in predicting the cut point of CORD BILIRUBIN total. The Logistic regression results are tabulated in table-12.

Table-12: Logistic regression on GA,SEX, ABO, Rh and CORD BILIRUBIN Total.

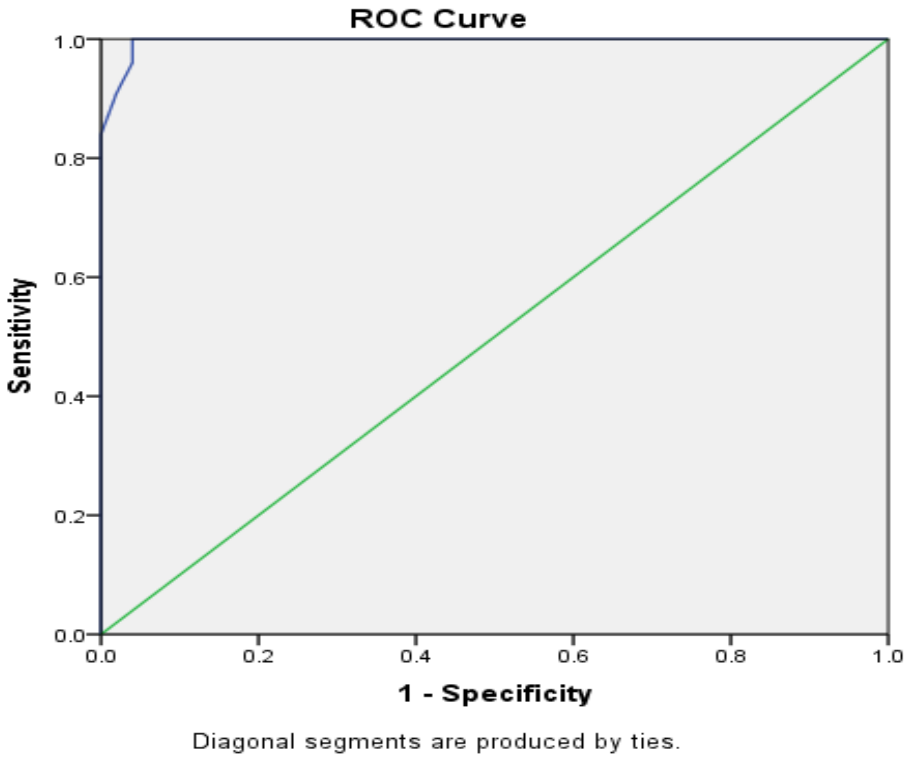
Variable	B	SE	Wald	df	P	EXP (B)
GA	2.609	1.919	1.849	1	0.174	13.587
Sex (M)	3.217	5.367	0.359	1	0.549	24.949
ABO	-2.149	1.788	1.444	1	0.229	4.117
Rh	-19.122	5191.355	0.000	1	.093	0.000
CORD BILIRUBIN Tot	11.447	4.342	6.949	1	.008	93591.7
Constant	-126.134	76.972	2.685	1	.101	.000

In the above table -12 Except CORD BILIRUBIN total all variables are not statistically significant (P>0.05). Hence the logistic regression was applied only for CORD BILIRUBIN total with NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA.

Table-13:Logistic Regression on CORD BILIRUBIN Total

Variable	B	SE	Wald	df	P	EXP (B)
CORD BILIRUBIN Total	9.197	2.950	9.716	1	.002	9864.989
Constant	-28.666	9.616	8.888	1	.003	0.000

Fig-2: Roc Curve for total CORD BILIRUBIN cut point.



Area Under the Curve

Test Result Variable(s): CORD BILIRUBINTOTAL

Area	Std. Error[a]	Asymptotic Sig.	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.996	.003	.000	.990	1.000

The curve estimation was done statistically significant (P<0.00). The diagonal segment would give the predictive value of CORD BILIRUBIN Total for HYPERBILIRUBINEMIA.

Table-14: Predictive value of HYPERBILIRUBINEMIA:

CORD BILIRUBIN Level	HYPERBILIRUBINEMIA	NHYPERBILIRUBINEMIA	Sensitivity	Specificity	PPV	NPV	Youden Index
≥3.25	99	2	96.0%	96.0%	98.0	97.9	0.959
<3.25	1	48					
Total	100	50					

The above table -13 states the cut off value is 3.25 of CORD BILIRUBIN Total. At this point the sensitivity is 96.0% and specificity is 96.0%and the Youden index is highest (0.959).

Table-15.Comparison of Total Serum Biliriubin (SBR) in different days:

Day	n	Mean	SD	Min	Max	'F'	Significance
0	50	2.3	0.3	1.9	3.5	177.38	Significant with all days
1st	34	4.4	0.5	3.2	5.6		Significant with all days
2nd	41	3.9	0.4	3.2	4.8		Significant with 0&1st day. 2nd, 3rd and 4th days were not significant.
3rd	20	3.6	0.3	3.2	4.3		
4th	5	3.7	0.7	3.3	3.9		
Total	150	3.4	0.9	1.9	5.6		

The SBR total of NONHYPERBILIRUBINEMIA was statistically significantly lesser than the other days. Similarly, the 1st day HYPERBILIRUBINEMIA total SBR was significantly greater than the other days. The other three days total SBR were significantly lesser than the 1st day.

Table-16: Comparison of HYPERBILIRUBINEMIA, from 12th to 24th hour, 24th hour to PHOTOTHERAPY transfusion and 12th hour to PHOTOTHERAPY transfusion.

Pair	Pre		Post		Difference		't'	df	Signifi
	Mean	SD	Mean	SD	Mean	SD			
12th to 24th	12.1	9.0	10.2	1.1	1.9	9.1	2.068	99	P<0.05
24th hour to PHOTOTHERAPY	10.2	1.1	8.4	1.3	1.8	0.7	25.334	99	P<0.001
12th hour to PHOTOTHERAPY	12.1	9.0	8.4	1.3	3.7	9.1	4.007	99	P<0.001

The reduction of reductions of total SBR with in the HYPERBILIRUBINEMIA were statistically significant.

Table-17: Association between ABO incompatibilities with PHOTOTHERAPY transfusion:

ABO	PHOTOTHERAPY transfusion						χ^2	df	significance
	No	%	Yes	%	Total	%			
No	93	67.4	11	91.6	104	69.3	2.025	1	P>0.05
Yes	45	32.6	1	8.4	46	30.7			
Total	138	100.0	12	100.0	150	100.0			

The above table shows that there was no significant association between ABO Incompatibilities and PHOTOTHERAPY transfusion (P>0.05)

Table-18: Association between Rh incompatibilities with

PHOTOTHERAPY transfusion:

Rh	PHOTOTHERAPY transfusion						χ^2	df	significance
	No	%	Yes	%	Total	%			
No	122	88.4	1	8.3	123	82.0	42.685	1	P<0.001
Yes	16	11.6	11	91.7	27	18.0			
Total	138	100.0	12	100.0	150	100.0			

The above table -17 shows the association between Rh incompatibilities with PHOTOTHERAPY transfusion. The results revealed that the PHOTOTHERAPY transfusion very strongly associated with Rh incompatibilities (P<0.001).

Table-19:Total SBR of HYPERBILIRUBINEMIA in difference days:

Day	n	Total SBR		Range
		Mean	SD	
1st	100	8.8	2.6	6.5-15.2
2nd	66	11.3	1.0	9.4-13.8
3rd	25	12.3	0.7	11.4-13.8
4th	5	13.5	0.5	13.1-14.2

The mean total SBR was in increasing trend.

DISCUSSION

Neonatal hyperbilirubinemia is one of the most common clinical entity that we come across with newborns. Mostly 85% of term and pre term newborns develop jaundice. Prediction of neonatal jaundice is more important as severe jaundice (Kernicterus) can occur in a healthy term baby with no clinically apparent jaundice in 24 hours. In this study we have arrived to a predictive value of cord bilirubin for neonatal jaundice in 1st week of life.

Our study population contains 150 newborns in which 100 newborns developed jaundiced and 50 newborns have no jaundice. Out which 37.3% were male baby and 62.7% were female baby. In this study we found that HYPERBILIRUBINEMIA did not have any significant association with the gender of the baby (P value more than 0.05). In one study among preterm group mean CORD BILIRUBIN was significant in males compared to female babies. In full term group mean CORD BILIRUBIN among male and female babies have no statistical significance. These results matched with Amar et al, Rostami and Mehrabi whereas it did not match with Ruby et al.

In this study the mean gestational age is 36.5 ± 1 and found this variable did not differ significantly. Mathias et al found that mean CORD BILIRUBIN is negatively correlated with gestational age.

In this study the mean birth weight is 26 ± 0.1 was found that this variable did not differ significantly (P value more than 0.05).

Adelia et al and Canceicao et al found that there was no significant correlation between total CORD BILIRUBIN and birth weight (≥ 3.5 kg). Preterm and low birth weight newborns are more likely to develop jaundice as the liver is immature with low UDPGT with decreased liver excretion.

In this study mode of delivery was 40% LABOUR NATURAL and 60% was CAESAREAN SECTION. Test of significance state that P more than 0.05 i.e., there was no statistical significance between CORD BILIRUBIN and mode of delivery. These results matched with Amar, Rostami and Mehrabi et al.

In this study ABO incompatibility requiring PHOTOTHERAPY is present in 30% and non ABO constitutes 69.3% showed that there was no statistically significant association between ABO incompatibility and PHOTOTHERAPY (P more than 0.05). In one study mean CORD BILIRUBIN for newborns who had PHOTOTHERAPY was 2.19 ± 0.24 mg/dL which is highly significant than those who didn't receive it (1.49 ± 0.34 mg/dL). This study matched with Adelia, Canceicao et al.

In this study about 18% of newborns with Rh incompatibility underwent PHOTOTHERAPY whereas 82% of newborns without Rh incompatibility underwent PHOTOTHERAPY. P value less than 0.001 indicates that PHOTOTHERAPY is very strongly associated with Rh incompatibility.

In this study the mean CORD BILIRUBIN was significantly higher among ABO +ve newborns compared to ABO -ve ones. The mean CORD BILIRUBIN is found to be greater in Rh than in ABO incompatible groups.

These results didn't agree with Adelia and Canceicao which showed that there is no significant difference in CORD BILIRUBIN between newborns with and without blood group incompatibility.

In this study the mean age of mother and newborn with no hyperbilirubinemia is 25.9 ± 2.1 years, whereas on the other hand with hyperbilirubinemia is 27.2 ± 4.1 years. The difference between the above mean was statistically significant with P less than 0.05.

In this study the mother with blood groups A-ve, B-ve, O-ve, O+ve were associated with HYPERBILIRUBINEMIA and A+ve, B+ve were not statistically associated with HYPERBILIRUBINEMIA (P value less than 0.001). Neonatal blood groups were not statistically significantly associated with HYPERBILIRUBINEMIA and NHYPERBILIRUBINEMIA.

In this study mean Hb of NONHYPERBILIRUBINEMIA was 14.2 ± 2.1 gm/dL and HYPERBILIRUBINEMIA was 15.6 ± 0.7 gm/dL. The difference between them showed a very high statistical significance.

In this study mean CORD BILIRUBIN between newborns with NONHYPERBILIRUBINEMIA and HYPERBILIRUBINEMIA were 2.3 ± 0.3 and 4.0 ± 0.5 respectively. The difference them proved statistical significance (P value less than 0.001).

In this study the relation between total CORD BILIRUBIN and SBR were statistically high significant. Logistic regression was applied only for CORD BILIRUBIN with NONHYPERBILIRUBINEMIA and

HYPERBILIRUBINEMIA because all other variables like GA, Sex, incompatibility were not statistically significant.

In this study CORD BILIRUBIN cut off point is calculated by ROC curve. The diagonal segment of this curve gives the predictive value of CORD BILIRUBIN as 3.25 mg/dL. At this point sensitivity and specificity were 96% with youden index being 0.959 (Highest).

Rudy et al determined this value using ROC as 2.54 mg/dL having high sensitivity and specificity. Amar et al's value was more than 2 mg/dL which had highest specificity and this critical bilirubin level had a very high NPV and fairly low PPV. Rostami and Mehrabi's value more than 3 mg/dL states that it was not a useful predictor for jaundice. Rataj et al showed that critical level in CORD BILIRUBIN $\geq$ 2.5 mg/dL had probability of 89% for the development of significant jaundice in new borns.

SUMMARY

This study was conducted to find out the predictive (Cut off) value of umbilical cord bilirubin in identifying neonatal hyperbilirubinemia in healthy term newborns. This study was carried out on 150 newborns in Tirunelveli Medical College Hospital. Babies were chosen based on inclusion and exclusion criteria. This study group was followed up clinically and with laboratory parameters - CORD BILIRUBIN was measured at birth followed up using TCB to find newborns developing jaundice in 1st week of life. Based on this the treatment modalities has been planned.

RESULTS OF STUDY:

Female babies were found to be higher i.e.,62.7% when compared to male babies 37.3%. Mean gestational age being 36.5± 0.1. Mean birth weight being 2.6 ± 0.1. There was no statistically significant association exist between CORD BILIRUBIN and mode of delivery. There was no statistically significant association exist between ABO incompatibility and PHOTOTHERAPY. Rh incompatibility is statistically significantly associated with PHOTOTHERAPY. Mean total CORD BILIRUBIN is more in Rh incompatibility (4.23) when compared to ABO incompatibility (3.86). Mean difference between age of mother with HYPERBILIRUBINEMIA and NONHYPERBILIRUBINEMIA is statistically significant. Mothers blood group has statistical signification with HYPERBILIRUBINEMIA and NONHYPERBILIRUBINEMIA but it was not seen in babies blood group. The mean Hb of HYPERBILIRUBINEMIA and

NONHYPERBILIRUBINEMIA is 15.6 ± 0.7 and 14.2 ± 2 gm/dL respectively and difference between them was statistically significant. The mean CORD BILIRUBIN between babies with HYPERBILIRUBINEMIA and NONHYPERBILIRUBINEMIA was 4 ± 0.5 and 2.3 ± 0.3 mg/dL respectively and difference between them was statistically significant. The relationship between total CORD BILIRUBIN and SBR was statistically significant

CONCLUSION

Study concludes that the total CORD BILIRUBIN in healthy term newborns provides prediction for neonatal jaundice in 1st week of life. The cut off value being 3.25 with 96.0% of specificity and 96.0% sensitivity. It is also evident that the presence of incompatibility in newborns (ABO, Rh) was statistically significant for occurrence of high total CORD BILIRUBIN that indicates PHOTOTHERAPY treatment.

PROFOMA

BABY DETAILS

Name :

Age :

Sex :

Date of Birth :

Gestational age : (Term / Preterm)

Apgar : 1 min 5 min

MOTHER DETAILS

Age :

LMP : EDD :

Blood group :

Anti-D given / Not :

History of drug in take / Jaundice / Blood transfusion / Fever with Rash:

Family history of Jaundice / Gallstones:

Thyroid disorder :

Pervious Sibling with Jaundice:

RISK FACTORS

Asphyxia / Sepsis / incompatibility / Hypoglycaemia / Hypothermia

HISTORY

Day of onset of Jaundice

High coloured urine / Pale stools

Birth Trauma

EXAMINATION

Clinical level of Jaundice (Kramer's rule)/ TCB

Head to foot examination

- Cephalohematoma / Subgaleal bleed / Bruises
- Wide open AF/ PF
- Cataract / Chorioretinits / Cherry red spot
- features s/o Down's Syndrome
- Umbilical sepsis
- Petechie / Blue berry muffin spots
- Hepatosplenomegaly
- Cardiac Murmur presence

INVESTIGATION

Cord Blood bilirubin

Baby blood group / Mother blood group

Serum bilirubin (Total / Indirect / Direct)

Haemoglobin levels

Day of Jaundice

TREATMENT

Phototherapy

Day started

Day Stopped

Exchange transfusion

Discharge

BIBILIOGRAPHY

1. American Academy of Paediatrics, Subcommittee on Neonatal Hyperbilirubinemia. Neonatal jaundice and kernicterus. Paediatrics. 2001;108:763–5.
2. American Academy of Paediatrics, Jaundice section of Infant nutrition and development of gastrointestinal function. In RE Kleinman, ed., Paediatric Nutrition Handbook, 6th ed., 2009; 47-49.
3. Kamath BD, Jaundice. In SL Gardner et al., eds., Merenstein and Gardner's Handbook of Neonatal Intensive Care, 7th ed., 2011; 531–552.
4. Sarci S U, Serdar MA, Korkmaz A, et al. Incidence, course and prediction of hyperbilirubinemia in near term and term newborns. Paediatrics.2004;113: 775–780.
5. Kawade N, Onishi S. The perinatal and postnatal development of UDP-glucuronyl transferase activity towards bilirubin and the effect of premature birth on its activity in human liver. Biochem J. 1981;196: 257–260.
6. Bhutani VK, Johnson LH, Maisels MJ et al. Kernicterus; epidemiological strategies for its prevention through system based approach. J Perinatol.2004; 24: 650–662.
7. Newman TB, Escobar GJ, Gonzales VM, Armstrong MA, et al . Frequency of neonatal bilirubin testing and hyperbilirubinemia in a large health maintenance organisation. Paediatrics 1999; 101: 995–998.

8. Maisels MJ, Kring E. Length of stay, jaundice and hospital readmission. PediatriCaesarean Section.1998; 101:995–998.
9. Maisela MJ, Newman TB. Jaundice in full term and near term babies who leave the hospital within 36 hours: the paediatrician's nemesis. Clin Perinatol.1998: 25: 295–302
10. Geiger AM, Petitti DB, Yao JF Rehospitalisation for neonatal jaundice. Risk factors and outcomes. Paediatric Perinat Epidemiol.2001: 15: 352–358.
11. Newman TB, Liljestrand P, Escoar GJ. Infants with bilirubin levels of 30 mg/dl or more in a large managed care organisation. Paediatrics. 2003; 111; 1303–1311.
12. Chou SC, Palmer RH, Ezhuthachan S et al. Management of hyperbiliubinemia in newborns: measuring performance by using a bench making model. Paediatrics. 2003: 112: 1264–1273.
13. Lee HC, Madan A , Hematologic abnormalities and jaundice. In CD Rudolph et al., eds., Rudolph's PediatriCaesarean Section, 22nd ed., 2011; 226–233.
14. Behrman RE, Kliegman RM, Jenson HB, eds. Jaundice and hyperbilirubinemia in the newborn. Nelson Textbook of Paediatrics. 16th ed. Philadelphia: Saunders, 2000;511–28.
15. Melton K, Akinbi HT, Neonatal jaundice. Strategies to reduce bilirubin-induced complications. 1999;106:167–8,171–4,177–8.

16. Maisels MJ, Newman TB, Kernicterus in otherwise healthy, breast-fed term newborns. Paediatrics. 1995;964 1:730–3.

17. Newman TB, Maisels MJ, Evaluation and treatment of jaundice in the term newborn: a kinder, gentler approach. PediatriCaesarean Section. 1992;895 1:809–18.

18. Knudsen A, Ebbesen F, Cephalocaudal progression of jaundice in newborns admitted to neonatal intensive care units. Biol Neonate. 1997;71:357–61.

19. Moyer VA, Ahn C, Sneed S, Accuracy of clinical judgment in neonatal jaundice. Arch Paediatric Adolesc Med. 2000;154:391–4.

20. Gartner LM, Herrarias CT, Sebring RH. Practice patterns in neonatal hyperbilirubinemia. Paediatrics. 1998;1011 Phototherapy 1:25–31.

21. Bhutani VK, Johnson L, Sivieri EM, Predictive ability of a pre discharge hour-specific serum bilirubin for subsequent significant hyperbilirubinemia in healthy term and near-term newborns. Paediatrics'. 1999;103:6–14.

22. Yetman RJ, Parks DK, Huseby V, Mistry K, Garcia J. Rebound bilirubin levels in infants receiving phototherapy. J Pediatr. 1998;133:705–7.

23. Jackson JC. Adverse events associated with exchange transfusion in healthy and ill newborns. Paediatrics. 1997;99:E7.

24. Brown AK, Kim MH, Wu YK, Bryla DA, Efficacy of phototherapy in prevention and management of neonatal hyperbilirubinemia. Paediatrics'. 1985;752 Phototherapy 2:393–400.

25. Manning D, Todd P, Maxwell M, et al. Prospective surveillance study of severe hyperbilirubinaemia in the newborn in the UK and Ireland. Arch Dis Child Fetal Neonatal 2007;92:F342–F346.
26. Ebbesen F, Andersson C, Verder H, et al. Extreme hyperbilirubinaemia in term and near-term infants in Denmark. Acta Paediatrica. 2005;94:59–64.
27. Smitherman H, Stark AR, Bhutani VK, Early recognition of neonatal hyperbilirubinemia and its emergent management. Semin Fetal Neonatal Med. 2006;11:214–224.
28. Maisels MJ, Physiologic and pathologic jaundice: the conundrum of defining normal bilirubin levels in the newborn. Paediatrics. 2006;118:805–807.
29. Wu PY, Hodgman JE, Kirkpatrick BV, et al. Metabolic aspects of phototherapy. Paediatrics. 1985;75:427–433.
30. Maisels MJ, Gifford K, Normal serum bilirubin levels in the newborn and the effect of breast-feeding. PediatriCaesarean Section. 1986;78:837–843.
31. US Preventive Services, Agency for Healthcare Research and Quality. Screening of infants for hyperbilirubinemia to prevent chronic bilirubin encephalopathy: US Preventive Services Task Force recommendation statement. Paediatrics'. 2009;124:1172–1177.

32. Trikalinos TA, Chung M, Lau J, et al. Systematic review of screening for bilirubin encephalopathy in neonates. PediatriCaesarean Section. 2009;124:1162–1171.
33. Ip S, Chung M, Trikalinos T, et al. Screening for bilirubin encephalopathy. Agency for Healthcare Research and Quality. 2009;1.
34. Mishra S, Chawla D, Agarwal R, et al. Transcutaneous bilirubinometry reduces the need for blood sampling in neonates with visible jaundice. Acta Paediatr. 2009;98:1916–1919.
35. Dijk PH, Hulzebos CV. An evidence-based view on hyperbilirubinaemia. Acta Paediatr Suppl. 2012;101:3–10.
36. Thayyil S, Milligan DW. Single versus double volume exchange transfusion in jaundiced newborn infants. Cochrane Database Syst Rev. 2006;(4):CD004592.
37. Reddy P, Najundaswamy S, Mehta R, et al. Tin-mesoporphyrin in the treatment of severe hyperbilirubinemia in a very-low-birth-weight infant. J Perinatol. 2003;23:507–508.
38. Murphy, J F; Hughes I; Verrier Jones ER; Gaskell S; Pike AW (1981). Pregnanediols and breast-milk jaundice.1981; 56: 474–76.
39. Nadir S, Saleem F, Amin K, Mahmood K (2011). Rational use of phototherapy in the treatment of physiologic jaundice neonatorum (PDF). Journal of Pharmaceutical Sciences and Research (Journal of Pharmaceutical Sciences and Research) 3 (1).

40. Dobbs, R H; R J Cremer , Phototherapy. Archives of Disease in Childhood 1975; 833–836.
41. Stokowski LA, Fundamentals of phototherapy for neonatal jaundice. Adv Neonatal Care (6)2006; 303–12.
42. Colletti, JE; Kothari, S; Kothori, S; Jackson, DM; Kilgore, KP; Barringer, K, An emergency medicine approach to neonatal hyperbilirubinemia. Emerg. Med. Clin. North Am. **25** (4):2007;1117–35.
43. American Academy of Paediatrics. AAP Issues New Guidelines for Identifying and Managing Newborn Jaundice. Retrieved 4 July 2009.
44. Kiviahan C, Jams EJ, The natural history of neonatal jaundice, PediatriCaesarean Section, 1984; 74, 364–370.
45. Martin CR, Cloherty JP. Neonatal hyperbilirubinemia, In Manual of Neonatal Care. In Cloherty JP, Eichenwald EC, Stark AR. 5th edition, Philadelphia, Lippincott Williams & Wilkins 2004; 185–221.
46. Kramer LI, Advancement of dermal icterus in jaundiced newborn. Am J Dis Child 1969; 118,454–8.
47. Maisels MJ, Baltz RD, Bhutani V, et al. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Paediatrics 2004;114,297–316.
48. Maisels MJ, Jaundice, In Avery's Neonatology Pathophysiology & Management of the Newborn. Eds: MacDonald MG, Mullett MD, Seshia MMK, 6th editon; Lippincott Williams & Wilkins, Philadelphia, 2005; 775.

49. Abu-Ekteish, F, Daoud, A, Rimawi, H, Kakish, K, & Abu-Heija, A, Neonatal exchange transfusion: Ajordanian experience. Annals of Tropical Paediatrics,2000; 20(1), 57–60.

50. Agarwal, R Kaushal, M Aggarwal, R Paul, V. K., & Deorari, A. K. Early neonatal hyperbilirubinemia using first day serum bilirubin level. Indian Paediatrics,2002; 39(8), 724–730.

51. Ahlfors, C. E. Unbound bilirubin associated with kernicterus: A historical approach. Journal of PediatriCaesarean Section,2000;137(4), 540–544.

52. AlOtaibi, S. F., Blaser, S., & MacGregor, D. L. Neurological complications of kernicterus. Canadian Journal of Neurological Sciences, 2005;32(3), 311–315.

53. Alpay, F., Sarici, S. U., Tosuncuk, H. D., Serdar, M. A., Inanc, N., & Gokcay, E. The value of first-day bilirubin measurement in predicting the development of significant hyperbilirubinemia in healthy term newborns. PediatriCaesarean Section,2000; 106(2), E16.

54. Basu, K Das, P. K Bhattacharya, R & Bhowmik, P. K. A new look on neonatal jaundice. Journal of the Indian Medical Association,2002; 100(9), 556–560.

55. Bental, Y. A Shiff, Y Dorsht, N Litig, E Tuval, L & Mimouni, F. B. Bhutani-based nomograms for the prediction of significant hyperbilirubinaemia using transcutaneous measurements of bilirubin. Acta Paediatrica,2009; 98(12), 1902–1908.

56. Bhat, Y. R & Rao, A. Transcutaneous bilirubin in predicting hyperbilirubinemia in term neonates. Indian Journal of PediatriCaesarean Section,2008; 75(2), 119–123.

57. Bhutani, V. K Gourley, G. R Adler, S Kreamer, B Dalin, C & Johnson, L. H. Noninvasive measurement of total serum bilirubin in a multiracial predischarge newborn population to assess the risk of severe hyperbilirubinemia. PediatriCaesarean Section,2000; 106(2), E17.

58. Carbonell, X Botet, F Figueras, J & Riu-Godo, A. Prediction of hyperbilirubinemia in the healthy term newborn. Acta Paediatrica, 2001;90(2), 166–170.

59. Centres for Disease Control and Prevention (CDC), Kernicterus in full-term infants--united states, 1994-1998. MMWR - Morbidity & Mortality Weekly Report,2001; 50(23), 491–494.

60. Funato, M Tamai, H Shimada, S & Nakamura, H. Vigintiphobia, unbound bilirubin, and auditory brainstem responses. PediatriCaesarean Section,1994; 93(1), 50–53.

61. Hansen TW. Mechanisms of bilirubin toxicity: Clinical implications. Clin Perinatol 2002;29:765–778.

62. Newman TB, Maisels MJ. Evaluation and treatment of jaundice in the term newborn: A kinder, gentler approach. Paediatrics 1992;809–18.

63. Penn AA, Enzmann DR, Hahn JS, Stevenson DF. Kernicterus in a full term infant. Paediatrics 1994;1003–6.

64. Stevenson DK, Fanaroff AA, Maisels MJ, et al. Prediction of hyperbilirubinemia in near-term and term infants. Paediatrics 2001;108:31–9.

65. Johnson LH, Bhutani VK, Brown AK. System-based approach to management of neonatal jaundice and prevention of kernicterus. J Pediatr 2002;140:396–403.

66. Centres for Disease Control and Prevention (CDC). Kernicterus in full term infants – United States, 1994-1998; MMWR Morb Mortal Wkly Rep 2001;50:491–4.

67. Dennery PA, Seidman DS, Stevenson DK. Neonatal hyperbilirubinemia. N Engl J Med 2001;344:581–90.

68. Seidman DS, Ergaz Z, Paz I, et al. Predicting the risk of jaundice in full-term healthy newborns: A prospective population-based study. J Perinatol 1999;19(8 Phototherapy 1):564–7.

69. Sarici SU, Yurdakok M, Serdar MA, et al. An early (sixth-hour) serum bilirubin measurement is useful in predicting the development of significant hyperbilirubinemia and severe ABO hemolytic disease in a selective high-risk population of newborns with ABO incompatibility. PediatriCaesarean Section 2002;109:e53.

70. Carbonell X, Botet F, Figueras J, Riu-Godo A. Prediction of hyperbilirubinaemia in the healthy term newborn. Acta Paediatr 2001;90:166–70.

71. Madlon-Kay DJ. The clinical significance of ABO blood group incompatibility. Arch Fam Med 1993;2:285–7.

72. Rubaltelli FF, Gourley GR, Loskamp N, et al. Transcutaneous bilirubin measurement: A multicentre evaluation of a new device. PediatriCaesarean Section 2001;107:1264–71.

73. Brown AK, Kim MH, Wu PY, Bryla DA. Efficacy of phototherapy in prevention and management of neonatal hyperbilirubinemia. PediatriCaesarean Section 1985;75(2 Phototherapy 2):393-400.

Sl. No	Name	Date of Birth	Gestational Age	Sex	Maternalage in Years	Birth weight (in Kg)	Apgar		AGA /LBW	Parity
							1 Min	5Min		
1	B/O Saraswathy	03-03-2015	36	MCH	30	2.5	7/10	8/10	AGA	1
2	B/O Ganasundari	03-03-2015	36	MCH	30	2.8	8/10	9/10	AGA	1
3	B/O Petchiammal	03-03-2015	36	MCH	28	2.6	8/10	9/10	AGA	1
4	B/O Jasmine	03-03-2015	37	FCH	28	2.7	7/10	8/10	AGA	1
5	B/O Aysha Beevi	04-03-2015	37	MCH	32	2.9	7/10	8/10	AGA	1
6	B/O Selidipinol	04-03-2015	36	MCH	26	2.1	7/10	8/10	LBW	2
7	B/O Muthu Selvi	04-03-2015	37	MCH	30	2.6	7/10	8/10	AGA	2
8	B/O Selvaklakshmi	04-03-2015	36	MCH	28	2.3	8/10	9/10	LBW	1
9	B/O Muthu Selvi	04-03-2015	36	FCH	28	3	8/10	9/10	AGA	1
10	B/O Syed Ali Fathima	04-03-2015	37	FCH	31	2.5	7/10	8/10	AGA	1
11	B/O Victoria	04-03-2015	36	MCH	29	2.9	8/10	9/10	AGA	2
12	B/O Mariselvi	04-03-2015	36	MCH	26	2.5	8/10	9/10	AGA	2
13	B/O Thaiammal	04-03-2015	36	MCH	28	2.5	8/10	9/10	AGA	1
14	B/O Asanbath	04-03-2015	37	FCH	30	2.3	7/10	8/10	LBW	1
15	B/O ArunaDevi	06-03-2015	37	MCH	28	2.8	7/10	8/10	AGA	1
16	B/O Nerosha	06-03-2015	36	FCH	25	2.7	7/10	8/10	AGA	1
17	B/O Mupidathy	06-03-2015	37	MCH	28	2	7/10	8/10	LBW	1
18	B/O Lakshmi	06-03-2015	36	MCH	22	2.5	7/10	8/10	AGA	1
19	B/O Petchiammal	06-03-2015	36	MCH	26	2.8	7/10	8/10	AGA	2
20	B/O Gomathiammal	06-03-2015	36	MCH	30	2.9	7/10	8/10	AGA	2
21	B/O Kuparal	06-03-2015	37	FCH	25	2.2	7/10	8/10	LBW	1
22	B/O Murugaeswari	06-03-2015	37	FCH	26	3.1	7/10	8/10	AGA	1
23	B/O Malarvili	07-03-2015	37	FCH	29	2.8	7/10	8/10	AGA	2
24	B/O Savithiri	07-03-2015	37	MCH	22	3.2	7/10	8/10	AGA	2
25	B/O Sasikala	07-03-2015	37	MCH	22	2.8	7/10	8/10	AGA	1
26	B/O Natchiaa	07-03-2015	36	MCH	29	2.9	7/10	8/10	AGA	1
27	B/O Uma Maheshwari	07-03-2015	37	MCH	22	2.2	7/10	8/10	LBW	2
28	B/O Tharani	07-03-2015	37	MCH	26	3	7/10	8/10	AGA	1
29	B/O Lakshmi	07-03-2015	37	MCH	26	2.9	7/10	8/10	AGA	1
30	B/O Kavitha	07-03-2015	36	FCH	20	2.8	7/10	8/10	AGA	3
31	B/O Ishaki Thai	07-03-2015	36	FCH	20	2.2	7/10	8/10	LBW	3
32	B/O Muthulakshmi	07-03-2015	36	FCH	22	2.6	8/10	9/10	AGA	3
33	B/O Karpagavalli	07-03-2015	36	FCH	25	2.8	7/10	8/10	AGA	3
34	B/O Ganamathi	07-03-2015	36	FCH	24	2.6	7/10	8/10	AGA	1
35	B/O Buvaneswari	07-03-2015	37	FCH	20	2.3	7/10	8/10	LBW	3
36	B/O Sathya	07-03-2015	36	FCH	27	2.8	7/10	8/10	AGA	3
37	B/O Subammal	07-03-2015	36	FCH	23	2.7	7/10	8/10	AGA	2
38	B/O Maragathavalli	09-03-2015	37	FCH	22	2.1	7/10	8/10	LBW	3
39	B/O Lakshmi	09-03-2015	36	FCH	24	2.8	7/10	8/10	AGA	1
40	B/O Poomari	09-03-2015	37	MCH	22	2.6	8/10	9/10	AGA	1

Sl. No	Name	Date of Birth	Gestational Age	Sex	Maternalage in Years	Birth weight (in Kg)	Apgar		AGA /LBW	Parity
							1 Min	5Min		
41	B/O Subulakshmi	09-03-2015	36	MCH	26	2.9	7/10	8/10	AGA	1
42	B/O Rohini	09-03-2015	37	MCH	30	2.8	7/10	8/10	AGA	1
43	B/O Poornima	09-03-2015	36	FCH	32	3.1	7/10	8/10	AGA	2
44	B/O Banumathi	09-03-2015	36	FCH	29	2.2	7/10	8/10	AGA	1
45	B/O Anita	10-03-2015	36	MCH	26	2.5	7/10	8/10	AGA	1
46	B/O Lakshmi	11-03-2015	36	FCH	35	2.6	7/10	8/10	AGA	1
47	B/O Muthu Lakshmi	11-03-2015	36	FCH	30	2.6	7/10	8/10	AGA	1
48	B/O Kanagumari	11-03-2015	36	FCH	29	2.2	7/10	8/10	LBW	1
49	B/O Maha Lakshmi	11-03-2015	36	FCH	29	2.6	7/10	8/10	AGA	1
50	B/O Gandhimathi	11-03-2015	37	FCH	26	2.4	7/10	8/10	LBW	2
51	B/O Ramalakshmi	11-03-2015	37	FCH	25	2.8	7/10	8/10	AGA	1
52	B/O Sudha	11-03-2015	37	FCH	28	2.2	7/10	8/10	LBW	1
53	B/O Kavitha	12-03-2015	37	FCH	32	3	7/10	8/10	AGA	2
54	B/O Maheswari	13-03-2015	37	FCH	28	2.1	7/10	8/10	LBW	3
55	B/O Shankari	13-03-2015	37	FCH	24	2.8	7/10	8/10	AGA	2
56	B/O Uthami	13-03-2015	37	FCH	25	2.5	7/10	8/10	AGA	3
57	B/O Amutha	14-03-2015	36	MCH	30	2.8	7/10	8/10	AGA	1
58	B/O Murugeswari	14-03-2015	37	FCH	28	2.2	7/10	8/10	LBW	2
59	B/O Vanitha	14-03-2015	37	FCH	26	2.9	7/10	8/10	AGA	1
60	B/O Subalakshmi	14-03-2015	37	FCH	22	2.6	7/10	8/10	AGA	2
61	B/O Kalaiselvi	14-03-2015	37	FCH	23	2.2	7/10	8/10	LBW	1
62	B/O Rajalakshmi	14-03-2015	36	FCH	26	2.8	7/10	8/10	AGA	2
63	B/O Ramayi	14-03-2015	36	FCH	28	2.6	7/10	8/10	AGA	1
64	B/O Manoramma	14-03-2015	36	FCH	28	3	7/10	8/10	AGA	1
65	B/O Malathi	15-03-2015	36	FCH	24	2.3	7/10	8/10	LBW	2
66	B/O Mariammal	15-03-2015	36	FCH	30	2.6	7/10	8/10	AGA	1
67	B/O Valavanthal	15-03-2015	37	FCH	30	2.1	7/10	8/10	LBW	2
68	B/O Roilala	16-03-2015	37	FCH	32	3	7/10	8/10	AGA	2
69	B/O Ishaki Ammal	16-03-2015	37	FCH	30	2.9	7/10	8/10	AGA	1
70	B/O Manoharamma	16-03-2015	36	FCH	29	2.8	7/10	8/10	AGA	1
71	B/O Lakshmi	16-03-2015	36	FCH	25	2.1	7/10	8/10	LBW	2
72	B/O Ajmal	16-03-2015	37	FCH	22	2.8	7/10	8/10	AGA	1
73	B/O Hyarnisha	16-03-2015	37	FCH	26	2.5	7/10	8/10	AGA	1
74	B/O Lakshmi	16-03-2015	36	MCH	20	3	7/10	8/10	AGA	1
75	B/O Uma Maheshwari	17-03-2015	37	FCH	29	2.3	7/10	8/10	LBW	1
76	B/O Pappa	17-03-2015	37	MCH	30	2.7	7/10	8/10	AGA	2
77	B/O Mumtaj Banu	17-03-2015	36	MCH	25	2.6	7/10	8/10	AGA	1
78	B/O Perumal	18-03-2015	37	FCH	28	2.7	7/10	8/10	AGA	2
79	B/O Rukmani	18-03-2015	36	MCH	25	2.2	8/10	9/10	LBW	1
80	B/O Aabikar	18-03-2015	36	MCH	28	2.7	8/10	9/10	AGA	1
81	B/O Bagavathy	18-03-2015	37	MCH	26	3	7/10	8/10	AGA	1

Sl. No	Name	Date of Birth	Gestational Age	Sex	Maternalage in Years	Birth weight (in Kg)	Apgar		AGA /LBW	Parity
							1 Min	5Min		
82	B/O Muthu Kalayani	19-03-2015	37	FCH	30	2.5	7/10	8/10	AGA	2
83	B/O Murugavalli	19-03-2015	36	FCH	26	2.8	7/10	8/10	AGA	1
84	B/O Shanthi	19-03-2015	37	MCH	35	2.5	7/10	8/10	AGA	2
85	B/O Shanthini	20-03-2015	37	FCH	28	2.1	7/10	8/10	LBW	1
86	B/O Maheswari	21-03-2015	37	FCH	25	2.8	7/10	8/10	AGA	2
87	B/O Murugaselvi	21-03-2015	37	FCH	26	2.9	7/10	8/10	AGA	1
88	B/O Nagammal	24-03-2015	36	MCH	30	3	7/10	8/10	AGA	1
89	B/O Arumugalakshmi	25-03-2015	36	FCH	26	2.6	8/10	9/10	AGA	2
90	B/O Amudha	25-03-2015	36	FCH	23	2	8/10	9/10	LBW	1
91	B/O Veyilatchi	25-03-2015	37	FCH	23	2.7	7/10	8/10	AGA	3
92	B/O Devi	25-03-2015	37	FCH	32	2.7	7/10	8/10	AGA	1
93	B/O Chellamari	25-03-2015	37	FCH	26	2.5	7/10	8/10	AGA	1
94	B/O Lakshmi	26-03-2015	36	MCH	31	2.6	7/10	8/10	AGA	2
95	B/O Punitha	27-03-2015	37	FCH	38	2.8	8/10	9/10	AGA	2
96	B/O Mariammal	27-03-2015	37	FCH	27	2.4	7/10	8/10	LBW	1
97	B/O Subha	27-03-2015	37	FCH	28	2.9	7/10	8/10	AGA	1
98	B/O Pirakasi	28-03-2015	37	FCH	27	2.9	7/10	8/10	AGA	1
99	B/O Thanthondri	28-03-2015	36	MCH	30	3	7/10	8/10	AGA	1
100	B/O Gomathi	28-03-2015	36	MCH	27	2.1	7/10	8/10	LBW	1
101	B/O Mariammal	28-03-2015	37	MCH	29	3.1	7/10	8/10	AGA	2
102	B/O Banumathy	28-03-2015	37	MCH	25	3	7/10	8/10	AGA	2
103	B/O Usha	28-03-2015	37	MCH	26	2.2	7/10	8/10	LBW	1
104	B/O Andal	28-03-2015	37	MCH	22	2.5	7/10	8/10	AGA	1
105	B/O Thalavaiselvi	30-03-2015	36	FCH	25	2.6	7/10	8/10	AGA	3
106	B/O Sundarammal	30-03-2015	37	FCH	26	2.6	8/10	9/10	AGA	1
107	B/O Muthukani	30-03-2015	37	FCH	25	2	8/10	9/10	LBW	1
108	B/O Maya	31-03-2015	37	FCH	21	3	7/10	8/10	AGA	2
109	B/O Ponnammal	01-04-2015	37	MCH	41	2.8	7/10	8/10	AGA	3
110	B/O Vasanthi	01-04-2015	36	MCH	25	2.6	7/10	8/10	AGA	1
111	B/O Uma Maheshwari	01-04-2015	37	FCH	24	2.7	7/10	8/10	AGA	2
112	B/O Indhra	01-04-2015	37	FCH	22	2.4	7/10	8/10	LBW	1
113	B/O Viswarohini	02-04-2015	36	MCH	24	2.5	8/10	9/10	AGA	1
114	B/O Vishnavi	02-04-2015	37	MCH	22	2.8	8/10	9/10	AGA	1
115	B/O Navanithan	02-04-2015	37	MCH	26	2.7	8/10	9/10	AGA	1
116	B/O Thavasimani	02-04-2015	37	FCH	28	2.7	8/10	9/10	AGA	1
117	B/O Subarani	02-04-2015	36	FCH	27	2.9	7/10	8/10	AGA	2
118	B/O Sankarammal	02-04-2015	36	FCH	22	2.2	7/10	8/10	LBW	1
119	B/O Gayathri	02-04-2015	36	FCH	25	2.6	7/10	8/10	AGA	1
120	B/O Malini	02-04-2015	37	FCH	24	2.8	7/10	8/10	AGA	2
121	B/O Murugalakshmi	03-04-2015	37	FCH	22	2.6	7/10	8/10	AGA	1
122	B/O Adhira	03-04-2015	37	FCH	26	2.8	7/10	8/10	AGA	1
123	B/O Esakiammal	07-04-2015	36	FCH	32	2.5	7/10	8/10	AGA	2

Sl. No	Name	Date of Birth	Gestational Age	Sex	Maternalage in Years	Birth weight (in Kg)	Apgar		AGA /LBW	Parity
							1 Min	5Min		
124	B/O Princes Pani Mary	07-04-2015	36	MCH	29	2.3	7/10	8/10	LBW	2
125	B/O Sankarammal	07-04-2015	36	MCH	28	2.8	7/10	8/10	AGA	2
126	B/O Chella Mary	08-04-2015	37	FCH	29	2.7	8/10	9/10	AGA	1
127	B/O Kaliswari	12-04-2015	37	FCH	24	3	7/10	8/10	AGA	2
128	B/O Sudha	12-04-2015	37	FCH	26	2.6	7/10	8/10	AGA	1
129	B/O Nandini	22-04-2015	37	FCH	30	2.1	7/10	8/10	LBW	1
130	B/O Thamayanthi	22-04-2015	37	FCH	27	3	7/10	8/10	AGA	1
131	B/O Shanthi	29-04-2015	37	MCH	36	2.8	7/10	8/10	AGA	1
132	B/O Parameswari	02-05-2015	36	FCH	25	2.6	7/10	8/10	AGA	1
133	B/O Banu Priya	02-05-2015	36	FCH	27	2.4	7/10	8/10	LBW	1
134	B/O Muthulakshmi	02-05-2015	37	MCH	24	2.5	7/10	8/10	AGA	1
135	B/O Genetamary	03-05-2015	37	FCH	27	2.6	7/10	8/10	AGA	1
136	B/O Maharani	03-05-2015	37	FCH	23	2.9	7/10	8/10	AGA	1
137	B/O Thirumalai Selvi	06-05-2015	37	FCH	21	3	7/10	8/10	AGA	2
138	B/O Sankarammal	06-05-2015	36	FCH	31	2.5	7/10	8/10	AGA	1
139	B/O Saraswathy	06-05-2015	36	MCH	31	2.6	7/10	8/10	AGA	2
140	B/O Thangam	06-05-2015	36	MCH	26	2.2	7/10	8/10	LBW	1
141	B/O Shanmugasundhari	08-05-2015	37	FCH	30	2.7	7/10	8/10	AGA	1
142	B/O Kanmani	10-05-2015	37	FCH	28	2.9	7/10	8/10	AGA	1
143	B/O Mahalaxmi	10-05-2015	37	FCH	29	3	7/10	8/10	AGA	1
144	B/O Thanga Selvi	13-05-2015	36	FCH	32	2.9	7/10	8/10	AGA	1
145	B/O Siva Sankari	13-05-2015	37	MCH	23	3	8/10	9/10	AGA	1
146	B/O Mupidathy	13-05-2015	37	MCH	25	2.4	8/10	9/10	LBW	1
147	B/O Ganasevli	13-05-2015	37	MCH	23	2.6	8/10	9/10	AGA	1
148	B/O Mupidathy	13-05-2015	37	FCH	21	2.9	7/10	8/10	AGA	2
149	B/O Perutchi	19-05-2015	36	FCH	27	2.8	8/10	9/10	AGA	1
150	B/O KaniMaryal	19-05-2015	37	MCH	25	2.9	8/10	9/10	AGA	1

Sl. No	Mode of delivery	Indication for CS	Blood Group		Hb Level	Anti-D Given	Hyperbilirubinemia Days						
			Mother	Baby			1	2	3	4	5	6	7
1	LN		AB+	B+	16.1			Yes					
2	CS	PIH	O+	A+	15.8		Yes						
3	CS		A+	A+	14.1								
4	CS	PIH	A+	A+	15.9			Yes					
5	LN		B-	B+	16.3	No	Yes						
6	LN		O+	B+	14.6								
7	CS	CPD IN	O+	B+	15.9			Yes					
8	LN		O+	B+	17.1		Yes						
9	LN		O+	A+	14.5								
10	CS		O+	A+	14.5			Yes					
11	LN		O+	A+	14.6			Yes					
12	CS	Previous LSCS	A+	A+	14.3								
13	CS		B+	B+	14.7								
14	LN		O+	O+	15.1				Yes				
15	LN		B+	AB+	16.2			Yes					
16	CS	CPD IN	B+	O+	14.5								
17	LN		O+	B+	15.8			Yes					
18	LN		B-	B+	15.9	No	Yes						
19	CS	Previous LSCS	A+	O+	14.2								
20	CS	PIH	O-	B+	14.5	Yes		Yes					
21	LN		A-	A-	15.2			Yes					
22	LN		A+	O+	14.3								
23	LN		AB+	B+	15.6			Yes					
24	CS	Previous LSCS	O+	A+	14.9				Yes				
25	CS		O+	A+	16.1				Yes				
26	LN		AB+	O+	14.7								
27	CS	Previous LSCS	O+	B+	15.7		Yes						
28	CS		O+	A+	15.1		Yes						
29	CS	PIH	O+	O+	14.1								
30	LN		A-	O+	14.9	Yes			Yes				
31	LN		O+	B+	15.1		Yes						
32	LN		A+	A+	14.8								
33	CS	CPD IN LABOUR	O+	A+	14.5			Yes					
34	CS		O+	A+	14.6								
35	LN		O+	A+	16.1			Yes					
36	LN		O+	B+	15.8			Yes					
37	CS		O+	A+	14.4								
38	LN		B-	B+	15.9	No	Yes						
39	CS	PRIMI WITH CPD	O+	A+	15.6		Yes						
40	CS		O+	B+	14.8			Yes					

Sl. No	Mode of delivery	Indication for CS	Blood Group		Hb Level	Anti-D Given	Hyperbilirubinemia Days						
			Mother	Baby			1	2	3	4	5	6	7
41	CS		O+	B+	14.5								
42	LN		O+	O+	14.6				Yes				
43	LN		B-	B-	15.3			Yes					
44	LN		A+	B+	14.7								
45	LN		B+	B+	16.3			Yes					
46	CS	PIH	O+	AB+	16.1				Yes				
47	CS	PIH	O+	A+	16.6		Yes						
48	CS		B+	B+	14.9								
49	CS	PIH	O+	B+	15.9			Yes					
50	LN		B-	B+	16.4		Yes						
51	LN		A+	A+	14.1								
52	LN		O+	A+	14.5				Yes				
53	CS	Previous LSCS	A+	A+	14.6			Yes					
54	LN		A-	A-	14.6				Yes				
55	LN		B+	B+	14.8								
56	LN		O+	A+	14.8			Yes					
57	CS	SHORT PRIMI WITH CPD	B-	B+	16.2	Yes	Yes						
58	LN		O-	O+	16.8		Yes						
59	LN		O+	O+	14.2								
60	LN		O-	B+	16.1		Yes						
61	LN		A+	A+	14.3								
62	CS	Previous LSCS	O+	B+	14.8			Yes					
63	CS		O+	B+	14.5								
64	LN		O+	O+	15.1			Yes					
65	LN		O-	B+	15.9		Yes						
66	LN		O+	O+	14.6								
67	CS	Previous LSCS	O+	O+	14.8			Yes					
68	LN		B+	B+	15.1				Yes				
69	LN		B-	B+	16.3		Yes						
70	LN		B+	B+	14.3								
71	CS		O-	O+	16.4	Yes	Yes						
72	LN		B-	B+	16.5		Yes						
73	LN		A+	A+	14.8								
74	CS	CPD	O-	O-	14.8					Yes			
75	LN		B+	AB+	14.6			Yes					
76	LN		O-	A+	15.9	Yes	Yes						
77	LN		B+	B+	14..4								
78	LN		A-	A+	15.5	Yes	Yes						
79	CS	PIH	O-	O+	15.6		Yes						
80	CS	PIH	O-	B-	14.3								
81	LN		O+	B+	14.7			Yes					

Sl. No	Mode of delivery	Indication for CS	Blood Group		Hb Level	Anti-D Given	Hyperbilirubinemia Days						
			Mother	Baby			1	2	3	4	5	6	7
82	LN		B-	B+	16.7	Yes	Yes						
83	LN		O+	A+	14.6								
84	LN		A-	A+	16.1	Yes	Yes						
85	CS	PIH	O-	B+	15.9		Yes						
86	LN		AB+	AB+	14.9				Yes				
87	LN		A-	A-	14.9								
88	LN		AB+	A+	15.1				Yes				
89	LN		O-	A+	17.3	Yes	Yes						
90	LN		A+	A+	14.7								
91	LN		B+	AB+	14.8			Yes					
92	LN		B-	B-	15.4			Yes					
93	LN		B+	O+	14.3								
94	CS	CPD	O+	O+	15.3			Yes					
95	LN		B+	B+	14.9				Yes				
96	LN		O+	B+	15.1				Yes				
97	LN		AB+	B+	14.7								
98	CS	CPD	O+	A+	16.4			Yes					
99	LN		O+	B+	15.1			Yes					
100	LN		B+	B+	14.6								
101	LN		O+	B+	14.9				Yes				
102	CS	Previous LSCS	O+	A+	14.5				Yes				
103	CS		B+	B+	14.2								
104	CS		AB+	A+	14.3								
105	LN		B+	B+	15.2					Yes			
106	LN		B+	A+	15.1			Yes					
107	LN		A+	A+	14.5								
108	CS	Previous LSCS	O+	B+	16.2			Yes					
109	LN		O-	A+	16.8	Yes	Yes						
110	LN		A+	B+	14.8								
111	CS	CPD	AB+	AB+	15.2			Yes					
112	CS		B+	A+	14.1								
113	LN		O+	B+	14.9				Yes				
114	LN		O+	O+	14.5								
115	LN		A+	O+	14.3								
116	CS		O+	A+	15.7			Yes					
117	LN		B+	B+	14.6				Yes				
118	CS	PIH	AB+	AB+	14.9					Yes			
119	CS		O+	B+	14.8								
120	CS	CPD	O+	O+	16.1			Yes					
121	LN		B+	B+	15.2					Yes			
122	LN		AB+	B+	14.2								
123	LN		AB+	B+	16.3			Yes					

Sl. No	Mode of delivery	Indication for CS	Blood Group		Hb Level	Anti-D Given	Hyperbilirubinemia Days						
			Mother	Baby			1	2	3	4	5	6	7
124	CS	Previous LSCS	B+	B+	15.3			Yes					
125	CS		A+	A+	14.6								
126	LN		O+	O+	15.6			Yes					
127	CS	PIH	O-	B+	16.9	Yes	Yes						
128	CS		A+	O+	14.3								
129	LN		O+	B+	14.9				Yes				
130	LN		O+	B+	14.8								
131	LN		A-	A+	16.2		Yes						
132	CS	CPD	O+	B+	14.9			Yes					
133	CS		O-	O-	14.2								
134	LN		O+	A+	15.7		Yes						
135	LN		B+	B+	15.2				Yes				
136	LN		O+	B+	14.7								
137	CS	Previous LSCS	AB+	B+	15.8			Yes					
138	LN		B+	B+	15.9					Yes			
139	LN		B-	B+	17.5		Yes						
140	LN		A+	A+	14.8								
141	CS	CPD	A-	B+	14.9		Yes						
142	LN		A-	A+	15.7		Yes						
143	LN		B+	B+	14.3								
144	LN		A-	A+	16.8		Yes						
145	CS	PIH	O-	O+	15.5		Yes						
146	CS		A+	A+	14.1								
147	CS		B+	B+	14.5								
148	CS	Previous LSCS	B+	B+	16.1			Yes					
149	CS	CPD	A+	O+	15.1				Yes				
150	LN	PIH	B+	O+	14.9			Yes					

Sl. No	CBR T/ID	SBR T/ID Days						
		1	2	3	4	5	6	7
1	4.6/3.6	6.9/5.8	11.1/10.6					
2	4.6/4.0	8.2/7.6						
3	2.2/1.1	2.6/2.1						
4	3.7/3.4	7.1/6.3	11.5/10.6					
5	4.4/3.4	12.8/11.9						
6	2.4/2.0	2.9/2.6						
7	4.1/4.0	7.2/6.5	12.0/11.5					
8	5.3/4.3	9.5/8.8						
9	2.3/2.0	2.7/2.5						
10	3.2/2.4	7.4/6.8	12.5/11.7					
11	3.6/3.2	7.4/6.7	12.2/11.4					
12	2.3/1.6	2.5/1.9						
13	2.0/1.6	2.2/1.8						
14	3.9/3.1	6.5/5.8	10.2/9.5	13.6/12.8				
15	4.5/3.4	6.8/6.1	12.1/11.2					
16	2.5/2.0	2.7/2.2						
17	4.2/3.6	6.5/5.8	11.5/10.7					
18	4.2/3.1	12.5/11.9						
19	2/1.5	2.3/1.9						
20	3.8/2.1	6.9/6.1	12.2/11.4					
21	4.2/3.4	7.4/6.6	13.3/12.6					
22	2.0/1.4	2.3/1.8						
23	4.8/3.9	6.8/6.1	12.2/11.4					
24	3.6/2.6	7.0/6.3	10.1/9.6	13.5/12.7				
25	4.3/3.2	7.3/6.5	10.4/9.7	13.8/12.9				
26	2.5/2.0	2.7/2.0						
27	4.0/3.2	10.6/9.8						
28	3.9/3.1	11.2/10.8						
29	2.3/1.7	2.5/1.9						
30	3.2/2.7	7.2/6.6	10.8/9.9	13.8/12.9				
31	3.2/2.4	11.2/10.4						
32	2.4/1.9	2.5/2.0						
33	3.5/2.6	6.9/6.1	12.6/11.8					
34	2.0/1.4	2.3/1.6						
35	4.3/3.4	7.2/6.8	13.8/13.4					
36	3.7/3.4	6.9/5.9	12.4/11.6					
37	2.5/1.8	2.6/2.0						
38	4.6/4.1	13.2/12.7						
39	4.3/3.5	10.5/9.7						
40	3.2/2.4	7.1/6.3	12.2/11.4					

Sl. No	CBR T/ID	SBR T/ID Days						
		1	2	3	4	5	6	7
41	2.1/1.8	2.3/1.9						
42	3.6/2.2	6.8/6.1	10.4/9.6	13.0/12.3				
43	4.2/3.6	6.5/5.8	11.9/10.9					
44	2.3/1.9	2.5/2.0						
45	4.3/3.4	6.9/6.1	11.8/10.9					
46	4.1/3.6	7.1/6.6	10.5/9.6	12.6/11.8				
47	4.4/3.6	10.8/9.9						
48	2.6/2.1	2.9/2.0						
49	4.1/3.4	7.2/6.4	12.6/11.8					
50	4.4/3.6	13.3/12.5						
51	3.1/2.6	4.8/3.9						
52	3.3/2.9	7.3/6.5	10.8/9.9	13.6/12.8				
53	3.5/2.8	6.9/5.9	11.6/10.9					
54	3.5/2.6	7.3/6.4	10.2/9.4	13.1/12.8				
55	2.0/1.2	2.3/1.9						
56	3.5/2.9	7.2/6.5	12.1/11.3					
57	4.4/3.7	12.1/11.2						
58	5.4/4.8	15.2/14.2	12.8/12.1					
59	2.6/1.8	2.9/1.3						
60	4.2/3.3	14.5/13.7	12.7/11.8					
61	2.3/1.9	2.5/2.0						
62	3.3/2.7	6.8/6.1	11.8/10.9					
63	2.0/1.4	2.3/1.7						
64	3.6/2.7	7.1/6.5	12.1/11.7					
65	4.6/3.7	13.1/12.3						
66	2.3/1.9	2.4/2.0						
67	3.9/3.1	7.2/6.6	12.6/11.8					
68	3.6/3.1	7.4/6.5	10.6/9.8	13.6/12.8				
69	4.6/3.9	13.9/13.1						
70	2.4/1.8	2.6/2.0						
71	4.2/3.6	14.1/13.6	12.2/11.5					
72	4.3/3.6	13.9/13.3						
73	2.9/2.4	3.0/2.5						
74	3.6/2.8	7.1/6.3	10.2/9.4	12.3/11.5	14.2/13.4			
75	3.7/3.1	6.9/6.1	13.1/12.4					
76	4.3/3.6	14.5/13.7						
77	2.2/1.8	2.5/2.0						
78	4.2/3.6	11.5/10.7						
79	4.3/3.9	11.8/10.9						
80	2.3/2.0	2.5/1.9						
81	3.6/2.9	7.1/6.4	12.5/13.7					

Sl. No	CBR T/ID	SBR T/ID Days						
		1	2	3	4	5	6	7
82	4.5/3.6	10.9/9.9						
83	2.1/1.5	2.4/2.1						
84	4.1/3.6	10.1/9.2						
85	4.0/3.9	11.3/10.6						
86	3.5/2.9	6.9/6.2	9.8/9.1	12.9/12.1				
87	2.3/1.9	2.4/2.1						
88	3.5/2.9	6.9/6.1	10.1/9.3	12.0/11.4				
89	5.1/4.1	14.2/13.6	12.2/11.4					
90	2.1/1.3	2.4/1.7						
91	3.6/2.8	7.1/6.5	12.1/11.6					
92	4.0/3.1	6.9/6.1	11.6/10.8					
93	2.0/1.3	2.2/1.6						
94	3.8/2.9	6.8/5.9	10.1/9.4					
95	3.6/2.9	7.1/6.3	10.3/9.6	12.2/11.7				
96	3.7/3.1	6.8/5.9	9.4/8.9	11.9/11.1				
97	2.5/2.0	2.6/1.7						
98	4.2/3.2	6.9/5.9	10.8/9.9					
99	3.6/2.9	7.2/6.4	12.1/11.7					
100	2.6/1.9	2.7/2.1						
101	3.7/2.8	7.1/6.5	10.6/9.7	12.6/11.8				
102	3.5/2.8	6.9/6.1	10.2/9.4	12.2/11.6				
103	2.0/1.4	2.2/1.6						
104	2.4/1.7	2.5/1.9						
105	3.8/3.1	7.2/6.3	10.6/9.8	11.4/10.8	13.6/12.8			
106	3.8/2.9	7.3/6.5	12.4/11.8					
107	2.2/1.7	2.5/2.1						
108	4/3.2	7.1/6.4	12.8/11.9					
109	4.8/3.9	14.6/13.9						
110	2.1/1.7	2.3/1.9						
111	3.8/2.9	6.9/6.1	11.6/10.8					
112	2.2/1.8	2.4/1.9						
113	3.9/3.1	7.2/6.4	10.4/9.5	12.8/11.9				
114	2.0/1.6	2.6/1.9						
115	2.2/1.6	2.5/1.7						
116	4.3/3.8	7.2/6.4	12.3/11.7					
117	3.3/2.5	7.2/6.4	10.3/9.5	13.1/12.6				
118	3.8/3.3	6.9/6.1	10.2/9.4	11.9/10.9	13.3/12.5			
119	3.3/2.6	4.5/3.8						
120	4.0/3.2	6.9/6.2	11.1/10.6					
121	3.3/2.7	7.1/6.3	10.1/9.3	11.4/10.6	13.1/12.6			
122	2.4/2.2	2.5/2.3						
123	4.1/3.6	7.1/6.5	11.5/10.6					

Sl. No	CBR T/ID	SBR T/ID Days						
		1	2	3	4	5	6	7
124	3.6/2.8	7.0/6.2	11.3/10.5					
125	1.9/1.2	2.2/1.7						
126	3.8/2.9	7.2/6.3	11.6/10.8					
127	4.9/4.2	11.1/10.6						
128	2.5/1.3	2.6/1.6						
129	3.6/3.1	7.1/6.8	10.2/9.5	13.2/12.6				
130	2.2/1.5	2.4/1.7						
131	4.2/3.6	13.1/12.6						
132	3.7/3.3	6.9/6.1	11.1/10.8					
133	3.5/2.8	4.6/3.9						
134	4.1/3.8	12.1/11.6						
135	3.3/2.6	7.1/6.2	10.3/9.6	13.6/12.8				
136	2.6/2.0	2.8/2.2						
137	3.8/2.9	7.3/6.5	11.9/11.1					
138	3.9/2.9	7.1/6.6	10.4/9.6	11.8/10.9	13.1/12.6			
139	5.6/4.8	11.7/10.9						
140	2.2/1.6	2.4/1.9						
141	3.6/2.5	12.1/11.6						
142	4.2/3.6	11.2/10.8						
143	2.7/2.0	2.9/2.2						
144	4.5/3.6	14.1/13.6						
145	3.6/2.8	11.1/10.6						
146	2.4/1.7	2.6/1.9						
147	2.0/1.2	2.3/1.6						
148	4.0/3.0	7.3/6.5	12.1/11.6					
149	3.6/2.8	7.0/6.2	10.3/9.5	12.6/11.8				
150	3.8/3.0	6.9/6.1	11.4/10.8					

Sl. No	Phototheraphy Days							12th Hour SBR	24th Hour SBR	REPEAT SBR [last PT]	Exchange Transfusion	DISCHARGED	Death
	1	2	3	4	5	6	7						
1								10.2/9.6	9.1/8.6	8.3/7.6		YES	NILL
2								7.5/6.9	6.9/6.0	5.3/4.6		YES	
3												YES	
4								10.1/9.2	9.1/8.5	8.8/7.9		YES	
5								11.6/11.0	10.2/9.8	7.9/6.9	Yes	YES	
6												YES	
7								11.0/10.5	10.1/9.1	8.0/7.3		YES	
8								9.1/8.6	8.5/7.8	8.1/7.2		YES	
9												YES	
10								11.2/10.6	10.1/9.3	7.9/6.9		YES	
11								11.1/10.5	10.2/9.4	8.2/7.6		YES	
12												YES	
13												YES	
14								11.1/10.4	10.2/9.3	8.2/7.5		YES	
15								11.1/10.3	10.1/9.6	8.1/7.6		YES	
16												YES	
17								10.1/9.3	9.2/8.6	6.9/6.2		YES	
18								10.2/9.2	8.8/7.6	7.1/6.4		YES	
19												YES	
20								11.0/10.4	10.2/9.3	8.1/7.4		YES	
21								12.2/11.2	10.1/9.2	9.1/8.6		YES	
22												YES	
23								11.0/10.2	10.4/9.6	7.9/7.1		YES	
24								12.0/11.2	11.2/10.3	9.1/8.2		YES	
25								12.2/11.6	11.0/10.4	9.4/8.4		YES	
26												YES	
27								9.6/8.4	8.5/7.8	6.9/6.1		YES	
28								10.2/9.4	9.5/8.6	7.8/6.9		YES	
29												YES	
30								12.8/12.0	11.4/10.8	10.1/9.3		YES	
31								10.2/9.8	9.6/8.9	8.1/7.6		YES	
32												YES	
33								11.0/10.5	10.2/9.8	8.3/7.6		YES	
34												YES	
35								12.2/11.6	10.1/9.6	9.9/9.1		YES	
36								11.0/10.8	10.2/9.6	8.0/7.3		YES	
37												YES	
38								12.2/11.8	11.1/10.8	8.9/7.9	Yes	YES	
39								9.2/8.6	8.5/7.8	7.2/6.5		YES	
40								11.2/10.8	10.6/9.8	8.2/7.6		YES	

Sl. No	Phototheraphy Days							12th Hour SBR	24th Hour SBR	REPEAT SBR [last PT]	Exchange Transfusion	DISCHARGED	Death
	1	2	3	4	5	6	7						
41												YES	
42								12.2/11.6	10.1/9.5	9.6/8.8		YES	
43								10.9/10.1	9.2/8.6	8.6/7.8		YES	
44												YES	
45								10.8/10.1	9.2/8.6	8.1/7.5		YES	
46								11.6/10.8	10.2/9.5	8.9/7.9		YES	
47								9.2/8.6	8.3/7.6	6.9/5.9		YES	
48												YES	
49								11.2/10.6	10.8/9.9	9.1/8.2		YES	
50								12.6/11.4	11.2/10.8	9.6/8.8		YES	
51												YES	
52								12.1/11.2	11.2/10.9	9.9/9.1		YES	
53								10.2/9.6	9.8/9.0	8.2/7.6		YES	
54								12.2/11.8	11.1/11.0	9.8/8.9		YES	
55												YES	
56								11.0/10.5	10.4/9.8	8.6/7.9		YES	
57								11.1/10.8	10.1/9.6	8.4/7.6		YES	
58								14.0/13.6	13.1/12.6	11.1/10.5	Yes	YES	
59												YES	
60								13.2/12.6	12.9/12.2	11.0/10.6	Yes	YES	
61												YES	
62								10.2/9.6	9.6/9.2	7.9/6.9		YES	
63												YES	
64								11.1/10.8	10.4/9.5	8.4/7.6		YES	
65								12.2/11.9	11.0/10.5	9.6/8.9		YES	
66												YES	
67								11.2/10.8	10.2/9.8	8.6/7.9		YES	
68								12.4/11.8	11.3/10.8	9.2/8.6		YES	
69								12.4/11.9	11.6/10.8	10.9/10.1	Yes	YES	
70												YES	
71								13.0/12.4	12.4/11.8	11.2/10.6	Yes	YES	
72								12.6/11.5	11.9/11.0	10.5/9.2	Yes	YES	
73												YES	
74								13.2/12.5	12.6/12.0	11.2/10.7		YES	
75								12.6/11.8	11.5/10.9	10.8/9.9		YES	
76								13.2/12.8	12.6/11.8	11.3/10.6	Yes	YES	
77												YES	
78								10.2/9.6	9.3/8.9	8.1/7.6		YES	
79								10.1/9.8	9.2/8.9	8.3/7.7		YES	
80												YES	
81								11.2/10.5	10.6/9.9	8.6/7.7		YES	

Sl. No	Phototheraphy Days							12th Hour SBR	24th Hour SBR	REPEAT SBR [last PT]	Exchange Transfusion	DISCHARGED	Death
	1	2	3	4	5	6	7						
82								9.5/8.9	8.5/7.8	6.2/5.6		YES	
83												YES	
84								9.2/8.6	8.3/7.6	7.3/6.2		YES	
85								10.2/9.6	9.1/8.5	8.2/7.4		YES	
86								11.6/10.8	10.5/9.9	7.8/6.9		YES	
87												YES	
88								11.2/10.6	10.1/9.8	8.9/7.9		YES	
89								13.2/12.8	12.8/11.6	11.0/10.6	Yes	YES	
90												YES	
91								11.2/10.6	10.8/9.5	7.9/7.1		YES	
92								10.4/9.8	9.5/8.6	7.3/6.8		YES	
93												YES	
94								9.6/8.5	8.4/7.8	6.3/5.8		YES	
95								11.2/10.6	10.2/9.5	7.1/6.4		YES	
96								10.5/9.8	9.2/8.6	7.3/6.5		YES	
97												YES	
98								9.6/8.9	8.2/7.8	6.6/5.8		YES	
99								11.2/10.8	10.1/9.5	8.9/7.9		YES	
100												YES	
101								11.3/10.6	10.2/9.4	8.6/7.8		YES	
102								11.2/10.5	10.3/9.5	8.5/7.8		YES	
103												YES	
104												YES	
105								12.0/11.3	11.2/10.6	10.5/9.8		YES	
106								11.0/10.6	10.1/9.6	9.8/8.9		YES	
107												YES	
108								11.2/10.8	10.2/9.7	8.9/8.1		YES	
109								13.2/12.8	12.1/11.6	11.3/10.5	Yes	YES	
110												YES	
111								10.2/9.4	9.3/8.4	7.2/6.9		YES	
112												YES	
113								11.6/10.6	10.2/9.6	6.9/5.9		YES	
114												YES	
115												YES	
116								11.2/10.7	10.4/9.7	7.2/6.8		YES	
117								12.2/11.4	11.6/10.8	9.6/8.8		YES	
118								12.1/11.6	10.6/9.8	9.5/8.6		YES	
119												YES	
120								10.2/9.5	9.5/8.8	7.1/6.3		YES	
121								12.0/11.2	11.2/10.6	7.9/6.9		YES	
122												YES	
123								10.2/9.6	9.6/8.9	7.3/6.5		YES	

Sl. No	Phototheraphy Days							12th Hour SBR	24th Hour SBR	REPEAT SBR [last PT]	Exchange Transfusion	DISCHARGED	Death
	1	2	3	4	5	6	7						
124								10.1/9.5	9.5/8.9	7.2/6.5		YES	
125												YES	
126								10.2/9.4	9.2/8.8	6.3/5.5		YES	
127								10.1/9.3	9.1/8.6	6.6/5.8		YES	
128												YES	
129								12.2/11.8	11.0/10.9	9.1/8.5		YES	
130												YES	
131								12.1/11.8	11.2/10.8	8.9/7.9		YES	
132								10.2/9.6	9.6/8.5	7.6/6.9		YES	
133												YES	
134								11.2/10.8	10.6/9.5	8.3/7.6	Yes	YES	
135								12.3/11.3	11.0/10.5	8.2/7.5		YES	
136												YES	
137								10.6/10.0	9.2/8.6	7.6/6.9		YES	
138								12.2/11.8	11.1/10.8	9.2/8.5		YES	
139								10.6/9.4	9.5/8.6	7.3/6.5		YES	
140												YES	
141								11.1/10.5	10.6/9.5	8.1/7.4		YES	
142								10.3/9.6	9.4/8.6	7.3/6.5		YES	
143												YES	
144								13.9/12.9	12.6/11.9	10.0/9.2	Yes	YES	
145								10.6/9.8	9.1/8.5	6.9/5.9		YES	
146												YES	
147												YES	
148								11.2/10.9	10.6/9.5	7.6/6.8		YES	
149								11.1/10.8	10.2/9.6	8.3/7.6		YES	
150								10.3/9.6	9.6/8.5	6.9/6.1		YES	